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DISEASES OF THE LIVER, GALL-BLADDER AND BILE-DUCTS

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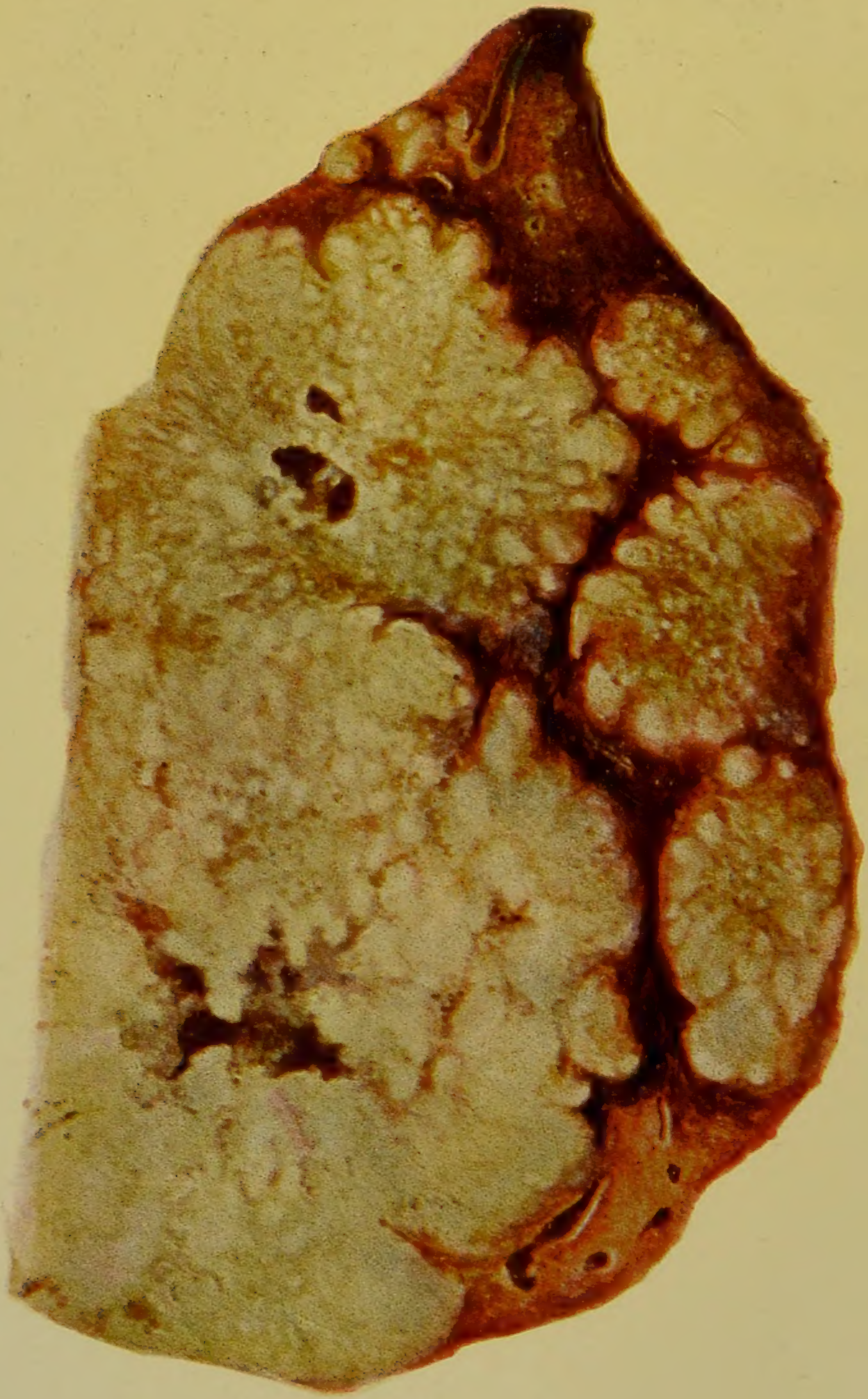

Growths in Liver Secondary to Carcinoma of the Rectum.

DISEASES

OF THE

LIVER, GALL-BLADDER AND BILE-DUCTS

BY

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PREFACE TO THE SECOND EDITION

THE work has been thoroughly revised; much new matter has been added, many alterations have been made, and condensation has been carried out as far as is compatible with a full consideration of the subject. I am specially indebted to Dr. A. J. Jex-Blake for reading the proofs, and to Dr. R. S. Trevor for generous help in many ways.

H. D. R.

September 1912.

PREFACE TO THE FIRST EDITION

During the past twelve years I have paid special attention to the diseases of the liver, both from the clinical and pathological points of view. Some of my observations have already appeared in various medical journals, Transactions of Medical Societies, and in articles on diseases of the liver in Vols. V and VI of the *Encyclopædia Medica* (in 1900).

Special Treatises often begin by dealing with the normal anatomy and physiology of the part concerned, but to be of any real value or use to the reader the anatomy and physiology of the liver must be dealt with in such detail as would seriously add to the bulk of the work, and I have therefore decided to omit any introductory chapter of this kind. I have done so with less regret as I believe that in most instances readers refer to textbooks or special monographs on the anatomy and physiology of the subject rather than to the introductory remarks at the commencement of a clinical treatise.

In the description of each disease attention is first directed to the underlying morbid changes, as without a grasp of these it is impossible to make a rational diagnosis, to treat the clinical manifestations in a satisfactory manner, or to give a reliable prognosis. Selected cases, for many of which I am indebted to my colleagues at St. George's Hospital, have been embodied in the text, especially in the parts dealing with the symptoms and clinical aspects. These cases and, in most instances, statistical details have been set in smaller print. The literature of the subject is enormous, and though no trouble has been spared, it is inevitable that omissions must exist. After full consideration I have decided to omit, in almost all cases, the bibliographies which had been prepared, and have only retained the references to authors actually quoted.

The illustrations are nearly all original, and I must cordially acknowledge my gratitude to many past students of St. George's Hospital for their help in this respect, especially to Dr. E. A. Wilson and Mr. Lawrence Jones (coloured plates, etc.), to Dr. H. Spitta and Mr. S. G. Penny (photomicrographs), and to Messrs. P. L. and S. P. Mummery, Dr. G. H. Goldsmith, and Mr. H. G. Drake Brockman. To Dr. H. Morley Fletcher, Professor S. Delépine, and Dr. T. Fisher I am indebted for permission to use or to copy figures which have already appeared elsewhere.

H. D. ROLLESTON.

November 1904.

Cysts with [illegible]

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Absence of the liver has been
found in monsters.

DISEASES OF THE LIVER

ANATOMICAL ABNORMALITIES

Abnormal Lobulation.—The liver has been found to be devoid of any division into lobes; more often there are many lobes so as to suggest a resemblance to the condition seen in some animals. This lobulation, which is not homologous to fetal lobulation of the kidneys, is more marked on the surface of the right lobe than on the left lobe. As many as 16 lobules have been found (Moser[1]). Whilst these lobules must be distinguished from the irregularities and hobnails of portal cirrhosis and from cicatrices manifestly syphilitic, it is probable that this lobulation is a pathological and not a morphological phenomenon. It may be due to syphilis, fetal peritonitis, or possibly to tuberculosis. Probably congenital syphilis is responsible for the change in many cases, but positive evidence to this effect is very often wanting. When lobulation of the liver is associated with peritoneal adhesions, it is possible that fetal peritonitis has so modified or interfered with the growth of the organ as to lead to fissuring and lobulation of its surface.

The view that lobulation of the liver might be caused by poisons manufactured by the *Bacillus tuberculosis* was put forward by Hanot,[2] who examined seven cases of lobulated livers in patients dying with tuberculosis, and considered that the lobulation was due to a coarse fibrosis induced by the toxins of tuberculosis (*vide* p. 347).

Alteration in the Relative Size of the Right and Left Lobes.—Occasionally the size of the right or of the left lobe is altered, with a corresponding change in the relative and absolute size of the other lobe. In rare instances the liver, though in its normal position, shews the lobulation seen in transposition of the viscera, the right lobe being small and the left large; or transposition of the lobes without *situs transversus*.

J. Davy[3] described a case in which the left lobe was twice the size of the right.

In some instances there is no manifest cause for this reversal of the normal lobulation of the liver, and it may be supposed that it depends on some alteration of the circulation in fetal or early life. In other instances the diminution in size of the right lobe is manifestly due to syphilitic cicatrices.

---

[1] Moser. *Med. Rec.*, N.Y., 1898, liii, 671.
[2] Hanot. *Gaz. des hôp.*, Paris, 1893, lxvi, 902.
[3] Davy, J. *Diseases of the Army*, p. 427, 1862.

Penrose[1] recorded this in acquired syphilis, and Lazarus-Barlow[2] in [illegible] ~~tardive~~ congenital syphilis.

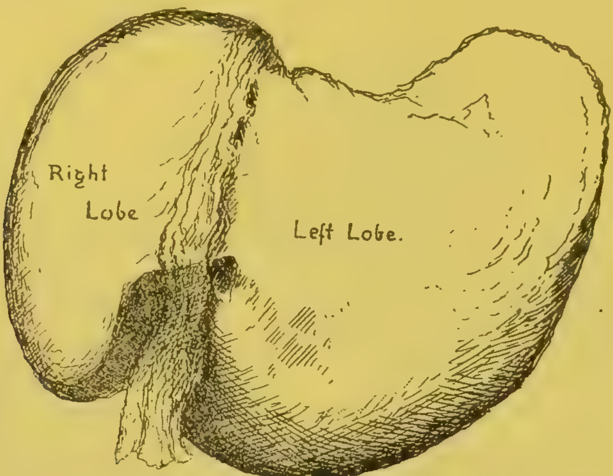
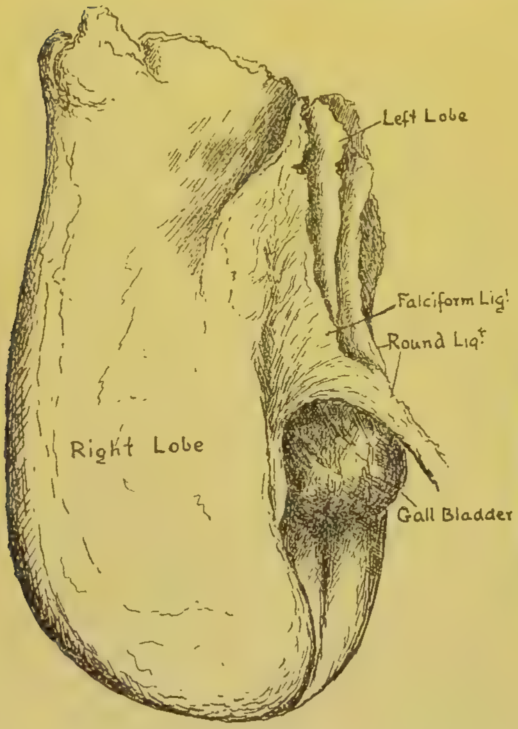


FIG. 1.—Liver with large left lobe and small right lobe; the gall-bladder was on the right lobe. (Drawn by Dr. G. H. Goldsmith.)



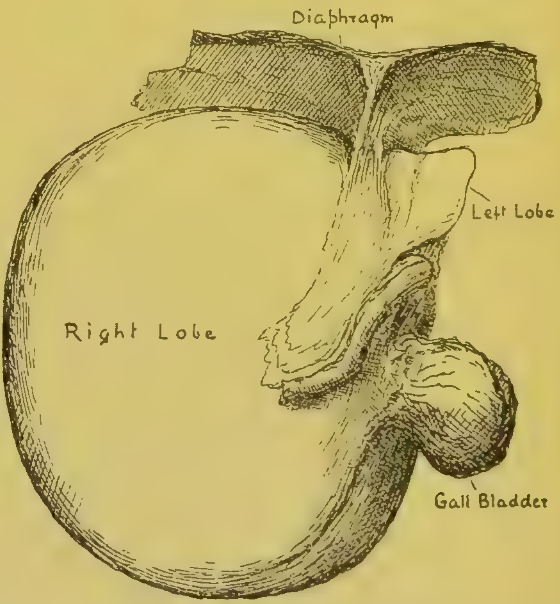


FIG. 2.—Extreme dwarfing of the left lobe of the liver. (Drawn by Dr. G. H. Goldsmith.)

FIG. 3.—Dwarfing of left lobe. The gall-bladder projects from the left margin of the liver and has its long axis in the transverse axis of the body. (Drawn by Dr. G. H. Goldsmith.)

---

[1] Penrose. *Trans. Path. Soc.*, Lond., 1889, xl, 133.

[2] Lazarus-Barlow. *Ibid.*, 1899, l, 158.

Dwarfing of one of the main lobes is occasionally seen, the other shewing compensatory hypertrophy. The left lobe is the one usually affected, and may be represented by a little flap of hepatic tissue. In these cases the left lateral ligament is correspondingly small and the falciform ligament arises from the left margin of the liver. The stomach is thus abnormally exposed, and the whole of the gall-bladder is visible from the front and projects from the left lateral margin of the liver. Owing to the disturbed relation of the lobes the gall-bladder may be so displaced as to lie with its long axis in the transverse axis of the body (*vide* Fig. 3).

There were 11 cases of dwarfing of one lobe (10 of the left lobe) among about 3000 necropsies at St. Bartholomew's Hospital (Herringham[1]).

There is seldom any very manifest reason why the left lobe should be so atrophied, and it may again be suggested that it is due to some disturbance of the circulation in fetal or early life. The change may be associated with other congenital defects.

Garrod[2] reported extreme dwarfing of the left lobe with lobulation of the right lobe of the liver and the presence of two instead of three aortic valves.

In other instances atrophy of the left lobe may be due to syphilis or to other pathological changes taking place later in life.

I have seen atrophy of the left lobe in a case in which a large intrahepatic calculus pressed upon the vessels entering the left lobe of the liver.

Accessory Lobes.—Small projections of liver substance—about the size of the terminal phalanx of the forefinger—which in miniature imitate the caudate lobe, are quite common and of no pathological significance. Their usual situation is the under surface of the right lobe, near the portal and longitudinal fissures. When markedly pedunculated, they may form "accessory livers." The Spigelian lobe is sometimes curiously pedunculated.

In the Anatomical Museum at Cambridge there is a cast of a pedunculated lobe attached to the left border of the left lobe by a pedicle composed of hepatic tissue. Lawrence and Nabarro[3] described an abnormal process from the left lobe of the liver in association with absence of the inferior vena cava in a female child aged fourteen weeks.

In rare instances large pedunculated tumours, apparently abnormalities and not obviously pathological in origin, may give rise to marked symptoms, such as pyloric obstruction.

An elongated process of the left lobe passed over the stomach and spleen and gave rise to gastric symptoms and signs suggesting a pancreatic cyst in a girl aged 16 years. Removal was followed by relief. Hammond[1] regarded it as congenital. A pedunculated tumour, measuring 3 × 2 inches, which caused pyloric obstruction, was successfully removed by Adams.[2]

---

[1] Herringham. *St. Barth. Hosp. Rep.*, 1905, xli, 19.

[2] Garrod, A. E. *Trans. Path. Soc.*, Lond., 1897, xlviii, 42.

[3] Lawrence and Nabarro. *Journ. Anat. and Physiol.*, 1901–2, xxxvi, 63.

Accessory Livers.—Isolated fragments of hepatic tissue or "rests" have been found in the suspensory ligament, but they are very rare, thus contrasting with the frequency of accessory suprarenals and of splenunculi.

Pepere[3] described innumerable small nodules of hepatic tissue or accessory livers scattered over the peritoneum and great omentum. One with a diameter of 7 cm. formed a solitary adenoma in the liver. Thirty-one years previously Wagner[4] recorded a number of nodules composed of liver cells in the falciform ligaments of two infants aged nine days and two months respectively. Chaillous[5] described a large accessory lobe attached to the anterior border of the liver, to the right of and close to the falciform ligament, on a level with the quadrate lobe; it was pear-shaped, like the gall-bladder. It was found in the body of an infant. Davy,[6] in the examination of a man aged twenty-three, dead of dysentery, found a small mass about the size of a hazel-nut, the structure of which appeared to be the same as that of the liver, attached to the concave surface of that organ by a delicate pedicle. Mahomet[7] described an accessory liver attached to the tip of the gall-bladder in a case of cirrhosis; the accessory liver was also cirrhotic. I have seen a somewhat similar condition in a lardaceous liver. Bland-Sutton[8] figures an accessory liver attached to the side of a gall-bladder.

Accessory livers may also be produced by atrophy of the liver cells in the pedicles of the minute accessory lobes so commonly seen on the under surface of the liver, with the result that a small peritoneal ligament unites the accessory liver to the main organ.

It seems possible that some detached lobes seen in adult life may be due to the effects of pressure or to atrophy of some of the liver tissue from interference with the blood-supply. Constriction lobes attached to the lower extremity of the right or more rarely the left lobe are described under the deformities of the liver due to tight lacing (*vide* p. 8). The following may be regarded as examples of atrophy of the intervening liver tissue inducing the appearances of an accessory lobe.

In a specimen in St. George's Hospital Museum the part of the liver representing the left lobe is completely separated for a distance of 3 inches from the rest of the liver; it was also attached by a kind of mesentery to the cardiac end of the stomach.[9] In a man aged twenty-four there was a vestige only of the left lobe, which was not continuous with the right lobe (Davy).

---

[1] Hammond, L. J. *Ann. Surg.*, 1905, xli, 31.

[2] Adams, J. A. *Glasgow Med. Journ.*, 1906, lxvi, 415.

[3] Pepere. *Arch. per le sc. med.*, 1902, xxvi, 117.

[4] Wagner. *Arch. der Heilk.*, 1861, i, 251.

[5] Chaillous. *Bull. Soc. Anat.*, Paris, 1898, lxvii, 572.

[6] Davy, J. *Diseases of the Army*, p. 428, 1862.

[7] Mahomet. *Trans. Path. Soc.*, Lond., 1877, xxviii, 147.

[8] Bland-Sutton. *Lancet*, Lond., 1907, i, 3.

[9] Series ix. 161A. See also Dickinson, W. H., *Trans. Path. Soc.*, Lond., 1867, xvii, 160.

Cullen figured hepatic tissue embedded inside an adrenal

Coroy reported a similar case

Cullen, T. S. Arch. Surg., Chicago, 1925, XI, 718.

Coroy, F. Compt. rend. Soc. biol., Par., 1922, LXXXVI, 695.

Atrophy may involve the base of attachment of the Spigelian (Davy[1]) or possibly of the caudate lobe and so lead to a pedunculated lobe.

It may be mentioned that between the peritoneal layers of the left lateral ligament the remains of a rudimentary lobe can be seen in the presence of hepatic and portal vessels, though the liver cells have disappeared. Occasionally small nodules of hepatic tissue are seen in this position (Tarozzi[2]).

Furrows on the surface of the liver not due to cicatrisation, as shewn by the healthy state of the peritoneum and underlying parenchyma, are fairly common, and were divided by Cruveilhier[3] in 1856 into (1) costal and (2) diaphragmatic. The costal grooves are seen on the lateral surface of the right lobe and their direction corresponds with that of the ribs. The diaphragmatic grooves are described on p. 10.

SOME POST-MORTEM APPEARANCES OF THE LIVER

A FEW words may be said about certain common post-mortem appearances of the liver, which will not be described elsewhere in this work.

Post-mortem Discoloration.—The surface of the liver where it has been in contact with the stomach or colon very commonly shews dark purple stains. These stains, which are produced after death and are quite superficial, are due to the action of gases, among them sulphuretted hydrogen, which diffuse through from the colon and stomach and meet with iron in the liver; as a result, some black compound like sulphide of iron is manufactured.

Irregular white areas on the surface of the liver are seen in cases of fever and various infections, and shew congestion and degenerative changes. This appearance was formerly thought to be merely due to mechanical pressure exerted after death in laying out the body.

Cloudy swelling is a very frequent, if not the commonest, change seen in the liver in routine post-mortem work. The liver is enlarged, heavier than normal, and looks as if it had been boiled, and has a duller, more opaque, and paler aspect than normal. These changes are due to cloudy swelling or parenchymatous inflammation of the liver cells set up by the toxins of numerous diseases. The changes which are shared by other organs, such as the kidneys and myocardium, are especially well seen in pneumonia. In this disease the enlargement of the liver may be very considerable. Long ago Bright[4] thought that the pneumonic lung materially depressed the liver, but it is clear that any increased hepatic dulness below the costal arch is mainly due to cloudy swelling and congestion (*vide* p. 19).

---

[1] Davy, J. *Diseases of the Army*, p. 427, 1862.

[2] Tarozzi. *Sperimentale*, 1904, lviii, 499.

[3] Cruveilhier. *Anatomie pathologique générale*, 1856, iii, 209.

[4] Bright, R. *Abdominal Tumours*, p. 255, 1860. New Sydenham Society.

Foaming Liver (*Synonym*: Emphysematous Liver).—The formation of gaseous cysts in the internal organs, formerly put down to putrefaction, was shewn by Welch and Nuttall,[1] in 1892, to be due to the *Bacillus aerogenes capsulatus.* The infection with this micro-organism is generally a secondary and terminal event; in other words, this bacillus follows in the wake of other pathogenetic bacteria and does not appear till the patient is moribund. To these general rules there are exceptions. Pure cultures of the *Bacillus aerogenes capsulatus* have been obtained by Pratt and Fulton,[2] and by Pakes and Bryant;[3] from a case of infective endocarditis, Gwyn[4] repeatedly isolated the micro-organism from the blood during life.

The micro-organism may be present and yet not give rise to any formation of gas (Gwyn, and Pratt and Fulton). ~~Though this infection may occur in the body during life, there is no evidence that it ever produces gas before death.~~ The micro-organism may give rise to necrosis of the cells of the liver and to purulent inflammation, as was shewn in Pratt's and Fulton's case of cholangitis with multiple abscesses in the liver.

The *Bacillus aerogenes capsulatus* is anaerobic, and must be distinguished from the bacillus of malignant oedema. It stains with Gram's method. It is very frequently present in the alimentary canal, and has been found to be widely distributed in nature. Other gas-producing bacteria must be taken into account; thus, the colon bacillus (Kanthack, Pakes and Bryant) and other members of the aerogenes group, such as *B. mucosus capsulatus* (W. T. Howard, Jr.[5]), have been described as giving rise to gaseous cysts. Welch, however, is rather sceptical about their claim to be regarded in this light.

The liver is the organ most frequently affected. In 23 cases tabulated by Pakes and Bryant, this organ was affected in 15.

Etiology.—*B. aerogenes capsulatus* is often found in association with other micro-organisms, which probably favour the development of *B. aerogenes capsulatus* by diminishing the bactericidal power of the blood, and also by reducing the resistance of the liver. J. H. Drysdale[6] found that hot weather is a factor in the production of foaming liver, but that this condition may occur in the absence of any of the ordinary signs of decomposition. It is found much more frequently when there has been recent loss of blood, which seems to favour the entrance of the micro-organisms into the blood or their development in it. Ulceration and injury of the intestines also favour the entrance of the micro-organisms, which are commonly present in the alimentary canal, into the tissues

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[1] Welch and Nuttall. *Bull. Johns Hopkins Hosp.*, Balt., 1892, iii, 81.

[2] Pratt and Fulton. *Boston Med. and Surg. Journ.*, 1900, cxlii, 599.

[3] Pakes and Bryant. *Guy's Hosp. Rep.*, 1900 (1897), liv, 283.

[4] Gwyn. *Bull. Johns Hopkins Hosp.*, 1899, x, 134.

[5] Howard, W. T., Jr. *Journ. Exper. Med.*, 1900–1, v, 139.

[6] Private communication.

Mc Neil will add a
few lines re
war experience

of the body. Incubation of livers, taken at random from the post-mortem room, did not give rise to the formation of gaseous cysts, but incubation of livers from cases of haemorrhage gave positive results proving that the micro-organisms had got into the organs.

It is generally recognised that the infection of the liver may take place either by the blood-stream or by the bile-ducts. Infection most commonly arrives by the portal vein, and is due to some lesion, such as ulceration, in the intestinal tract. The inlet of infection may be in other situations, such as the uterus or the urethra. Direct infection of the bile-ducts and gall-bladder from the intestine may occur. In such instances the bile-ducts and gall-bladder may contain gas either in their lumen or in the substance of their walls, especially in the submucous coat. Welch[1] met with five cases of interstitial emphysema of the bile-ducts or gall-bladder.

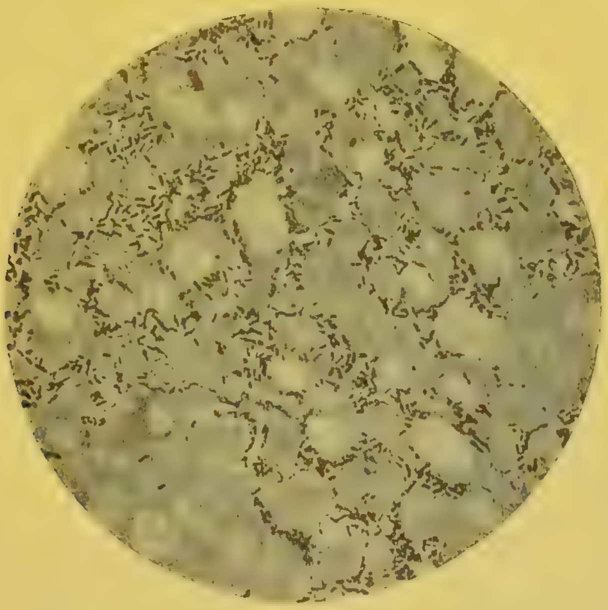

FIG. 4.—Photomicrograph of a foaming liver, shewing minute gaseous spaces lined by the *Bacillus aerogenes capsulatus.* × 1000. (By Dr. H. Spitta.)

Although the gas-producing micro-organisms may give rise to a septicaemia and be isolated from the blood, it is doubtful if formation of gas ever occurs in the living tissues. It may occur very rapidly after death, or, as shewn in emphysematous gangrene, in a part of the body that has lost its vitality. On reviewing the whole subject Welch is inclined to the view that gas is formed in the viscera before death. Clinically it has been thought that tympanites may be due to the formation of gas before death.

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[1] For a full résumé of the entire subject the reader should refer to Welch's Shattuck Lecture for 1900, "On Morbid Conditions caused by *Bacillus aerogenes capsulatus,*" *Johns Hopkins Hosp. Bull.*, Balt., 1900, xi, 185.

The liver is spongy from the presence of a number of small gas-containing cysts of various sizes, but mostly small. The organ is usually of a somewhat greyish colour from its inflated condition, which must not be mistaken for multiple cystic disease. This change may also be met with in other organs—the kidneys, brain, spleen, intestines, pancreas. Some recorded cases of widespread cystic change in the organs of the body may have been of this nature.

ACQUIRED DEFORMITIES

Tight-laced Liver (*Synonym*: Corset Liver).—Modifications in the shape of the liver due to tight lacing, corsets, and belts are of course common in women, but considerable deformity may be produced in men by the pressure of a tight belt or strap. The effect of tight lacing on the liver varies to a certain extent with fashion, or, in other words, with the position of the waist. Following Hertz,[1] who has studied the changes produced by tight lacing in great detail, the deformities of the liver may be divided into two main types, though mixed or transitional forms may occur.

I. The liver is flattened and elongated from above downwards so that the upper or diaphragmatic surface is diminished while the anterior is increased. From its larger size the right lobe naturally shews the change more than the left. The liver thus forms a flap which covers the abdominal viscera, though occasionally coils of intestine may pass in front of it. Where it passes over the right kidney the elongated right lobe shews atrophy of the hepatic substance and thickening of the capsule, which is opaque and forms a hinge-like ligament between the main part of the right lobe above and the constricted lower portion. This lobe is variously termed partial hepatoptosis, constriction lobe, or the sustentacular formation of the right lobe (Hertz). The constriction furrow is produced by the pressure of the corset in front and the resistance of the kidney behind. The constriction lobe tapers to a point so that the shape of the liver, as seen from the front, is that of a right-angled triangle with the apex downwards. This lobe is often associated with an unduly movable or floating kidney (Keith [2]).

Clinically there is a close resemblance between these constriction lobes of the right lobe and the tongue-shaped or Riedel's lobe, originally described as occurring in special association with gall-stones. The left lobe is prolonged downwards in the same manner, though to a less marked degree, and may even have a constriction lobe attached to it. It has been thought that in the latter event the symptoms were much more marked than in cases in which the ordinary tongue-like lobes from the

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[1] Hertz, P. *Abnormitäten in der Lage und Form der Bauchorgane bei dem erwachsenen Weibe*, Berlin, 1894.

[2] Keith, A. *Lancet*, 1903, i, 711.

right lobe were present. A tongue-like lobe arising from the left lobe of the liver would tend to exert pressure on the pylorus, duodenum, pancreas, and nerve plexuses which are supported behind by the spine, while on the right side no important viscera or structures would suffer, though the lobe would probably be in contact with the right kidney.

From impaired nutrition and diminished resistance the constriction lobe may be more markedly affected by morbid changes than the rest of the liver, or may even be the only part affected. Thus, fibrosis or gummatous change may be confined to a constriction lobe, or secondary new-growth may be wholly or chiefly limited to the pendulous lobe.

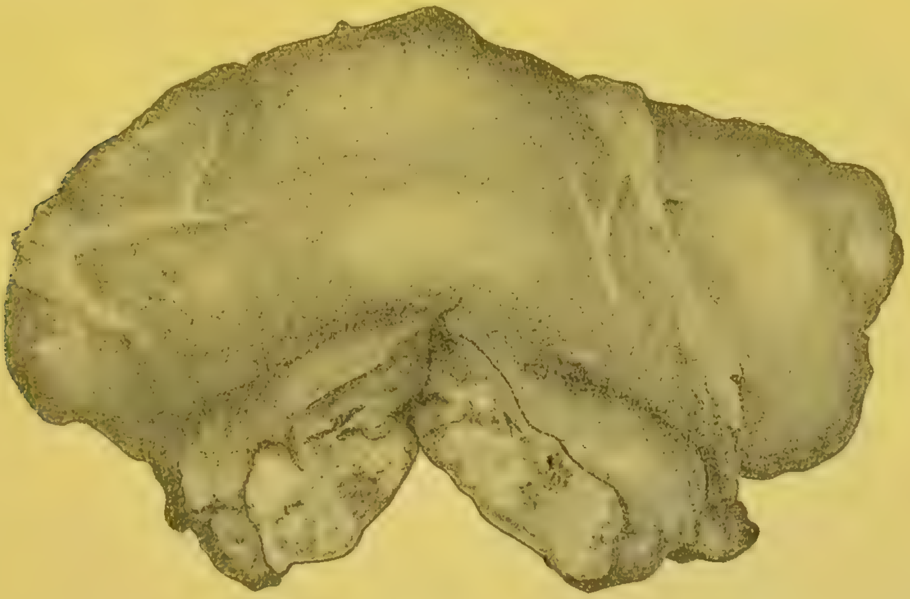

FIG. 5.—Constriction lobe attached to the right lobe of the liver, divided and its halves separated so as to display large secondary carcinomatous nodules in its substance. From case described in the text. (Drawn by L. Jones, M.S.)

In a case of carcinoma of the mamma in a woman who died of diabetes the constriction lobe contained large masses of secondary growth, whilst in the remainder of the liver there were only two small nodules (Fig. 5). Frerichs[1] figures a similar case.

On the other hand, the interference with the blood-supply may to some extent protect the constriction lobe against infection by the blood-stream.

In 1890 I examined after death the body of a man who died with carcinoma of the pylorus; the liver weighed nine pounds and, except the constriction lobe, attached to the right lobe, was full of secondary new-growth.

The whole of the liver, constriction lobe included, may be uniformly affected by cirrhosis (Fig. 6).

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[1] Frerichs. *Diseases of the Liver*, ii, 326, Transl. New Sydenham Soc., 1861.

II. In the second variety of tight-laced liver the organ is displaced upwards as a whole and lies high in the abdominal cavity. It is thicker above than below, and is curved and moulded over the spine so that the left lobe may touch or even overlap the spleen. Frerichs[1] figures such a case in which the left lobe of the liver and the spleen were firmly united to each other. The fossa for the inferior vena cava is exaggerated and deepened, while as the result of pressure the right kidney is displaced downwards and its lower end tilted forwards. The lower margin of the right lobe being compressed by the corset or belt, is atrophied in the transverse direction, and shews opacity and thickening of the capsule from local perihepatitis. A triangular constriction lobe may be found attached to the right lobe, but is not such a constant feature as in the previous form, and is never present in connexion with the left lobe. Besides being met with in women, this change in the liver may be met with in men who wear a tight belt.

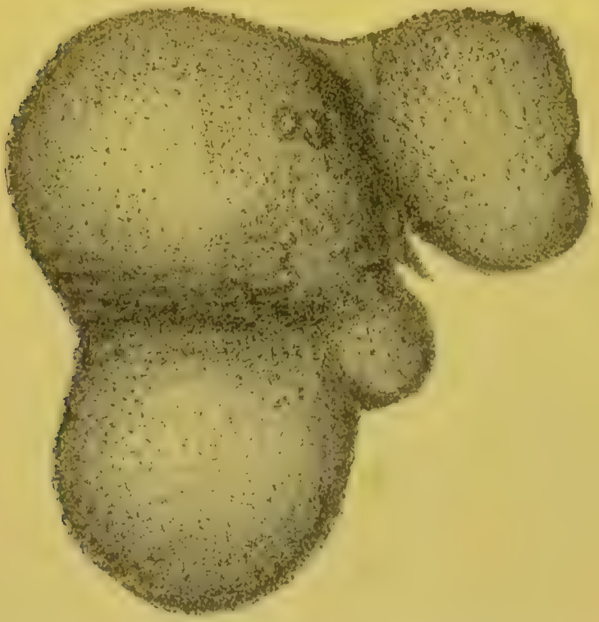

FIG. 6.—Tight-laced liver uniformly cirrhotic. There are two small cysts on the right lobe. From a woman forty-four years old who died with grave anaemia. The liver weighed forty-one ounces. (Drawn by P. L. Mummery, M.B., F.R.C.S.)

Diaphragmatic Sulci.—These are parallel grooves on the convexity of the right lobe, and in rare instances of the left lobe; there may be from one to six of these furrows; Moody[2] mentions a liver with nine. They run antero-posteriorly and are deeper posteriorly. It has been estimated that they occur in 7·3 per cent of all bodies (Adami and Nicholls[3]). They are eight times commoner in women than in men. They are usually associated with emphysema and bronchitis, and with evidence of tight lacing. Various explanations have been given (1) that they are due to circumferential pressure exerted in an upward direction by belts, and in some cases there is a transverse band of thickening near the anterior inferior margin of the liver (Weber[4]); (2) that they are due to the impress left by hypertrophied bundles of the diaphragm and

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[1] Frerichs. *Diseases of the Liver*, 1860, i, 41.
[2] Moody, R. O. *Johns Hopkins Hosp. Bull.*, 1905, xvi, 364.
[3] Adami and Nicholls. *Principles of Pathology*, 1911, ii, 460.
[4] Weber, F. P. *Trans. Path. Soc.*, 1897, xlviii, 113; 1900, li, 236.

That diaphragmatic sulci
are ever congenital is
denied by Collet and Trossat.

Collet et Trossat. Lyon méd., 1922, cxxxi, 467 are not due to pressure (Zahn[1]); the change in the diaphragm being the result of chronic diseases of the respiratory system, with which these furrows are frequently associated. In 58 cases from the Salpêtrière shewing these furrows there were only 5 in which the lungs were healthy (Séglas[2]). Very probably both these factors act together. It is improbable that they are due to pressure exerted by the ribs, inasmuch as the sulci do not correspond to the position of the ribs.

Bacaloglu[3] described a single deep furrow on the upper surface of the liver which had no relation to either the ribs or the diaphragm and was not the result of cicatrisation. He regarded it as directly due to the pressure of the corset.

Furrows of this kind have in rare instances been seen in fetuses, and are possibly due to disproportion between the size of the abdominal cavity and of the liver so that the latter becomes folded in the process of growth (Moody). They must be distinguished from those due to hereditary syphilis. ∧ The etiology of ~~these~~ diaphragmatic sulci is discussed in detail by Moody.

Cirrhosis may supervene in a liver the subject of tight lacing, and give rise to a uniform change (*vide* Fig. 6). Local changes, probably due to chronic venous engorgement and resulting in fibrosis, may occur in the portion of the liver below the tight-lacing constriction.

Clinical Features.—Tight-laced livers are often associated with dyspepsia, which may be partly due to the abdominal or gastric embarrassment produced by a tight corset. In other cases the symptoms are due to visceroptosis, which is often associated with tight lacing. Keith[4] has insisted on the importance of tight lacing as a cause of gall-stones, and symptoms of cholelithiasis are not uncommon in patients who have tight-laced livers. But in a very considerable proportion of patients with livers deformed by tight lacing no symptoms referable to that organ are present. A point of considerable interest about tight-laced or corset livers is that the constriction lobe may, when accidentally detected, be easily mistaken for something more important, such as a floating kidney, a tumour of the pylorus or transverse colon, a dilated gall-bladder, cysts of the pancreas or of the mesentery, or in extreme cases for a fibromyoma of the uterus, an ovarian tumour, or appendicitis. The connecting bridge between the constriction lobe and the main part of the liver, as has already been pointed out, is sometimes very thin, and may therefore give a resonant note on percussion, so that its continuity with the remainder of the organ is concealed.

Treatment.—As a rule, no active treatment for the constriction lobe is either required or justifiable; tight lacing should as far as possible be prevented, but this reform requires considerable tact. A straight-fronted corset should be substituted for one which tends to constrict the waist and depress the liver. A properly adjusted belt should be fitted to patients with enteroptosis and symptoms due to this cause. Dyspeptic and other associated symptoms should be carefully attended to and constipation prevented. In cases in which the constriction lobe is the seat of much pain it has been removed, but this can seldom be really necessary. In such cases Bötticher[1] considers that hepatopexy, or fixation of the constriction lobe, is a more satisfactory method of surgical treatment.

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[1] Zahn. *Rev. méd. de la Suisse Romande*, 1882, ii, 19.
[2] Séglas. *Progrès méd.*, Paris, 1886, 2. s., iii, 471.
[3] Bacaloglu. *Bull. Soc. anat.*, Paris, 1899, lxxiv, 67.
[4] Keith, A. *Lancet*, 1903, i, 639.

Tongue-like Lobes (*Synonyms*: Linguiform Lobe, Riedel's Lobe, Partial Hepatoptosis, Floating or Appendicular Lobe).—Although the term partial hepatoptosis has been employed, this condition is quite distinct from complete hepatoptosis or wandering liver, as there is no dropping of the organ as a whole. No doubt confusion between the two conditions has occurred; Glénard considers that the reputed greater incidence of wandering liver in women depends on the fact that some observers have erroneously described tongue-like lobes as wandering livers. These lobes, often spoken of as Riedel's, are really much the same as the constriction lobes just described (*vide* p. 8) in the account of the corset liver. It has been thought, especially by Riedel,[2] that the tongue-shaped lobes depend on disease of the gall-bladder, such as inflammation, gall-stones, distention, traction exerted by pericholecystic adhesions. But this explanation does not apply to all the cases.

Omit

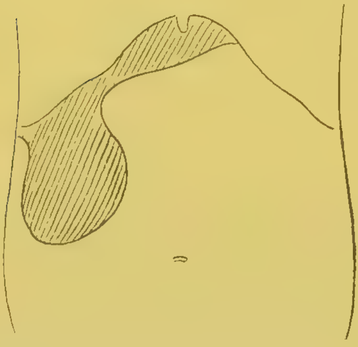

FIG. 7.—Riedel's tongue-like lobe. (After Dr. T. Fisher.)

It is possible that some of the cases of downward projection of the right lobe are connected with or depend on a congenital anatomical variation. This suggestion applies forcibly to cases in which this formation is found in babies or young children. As pointed out by Fisher,[3] there is a considerable amount of variability in the outline of the right lobe, and exaggeration of this might occur as an occasional abnormality without any determining irritation or traction on the part of the underlying gall-bladder. Possibly the formation of Riedel's lobe in some cases of cholelithiasis and its absence in other cases depend on variability in the outline of the right lobe of the liver.

Dr. Fisher has kindly sent me a drawing of a potential Riedel's lobe in a woman aged twenty-five whose gall-bladder was normal; I have observed a similar abnormal lobulation in the body of a man aged fifty-eight. If gall-

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[1] Bötticher. *Deutsche Ztschr. f. Chir.*, 1900, lvi, 252.
[2] Riedel. *Berlin. klin. Wchnschr.*, 1881, xxv, 577, 622.
[3] Fisher, T. *Bristol Med.-Chir. Journ.*, 1901, xix, 215.

Need to get
a fresh illustration

bladder disease arises in such cases, a Riedel's lobe would probably develop much more readily and rapidly than in a normal liver. McPhedran[1] described seven cases, and regarded the tongue-like lobes as developmental and not artificial. One of his cases was in a baby of eleven months. I have seen an infant four days old with numerous other abdominal malformations in which the gall-bladder lay on a lobe united to the right lobe of the liver by peritoneum and adherent to the umbilicus.

Tight lacing must also be taken into account, especially as, by displacing the fundus of the gall-bladder downwards, it may lead to kinking and occlusion of the cystic duct and so to obstruction to the outflow of the contents of the gall-bladder—a state of affairs which favours catarrhal inflammation and the production of gall-stones (Keith[2]).

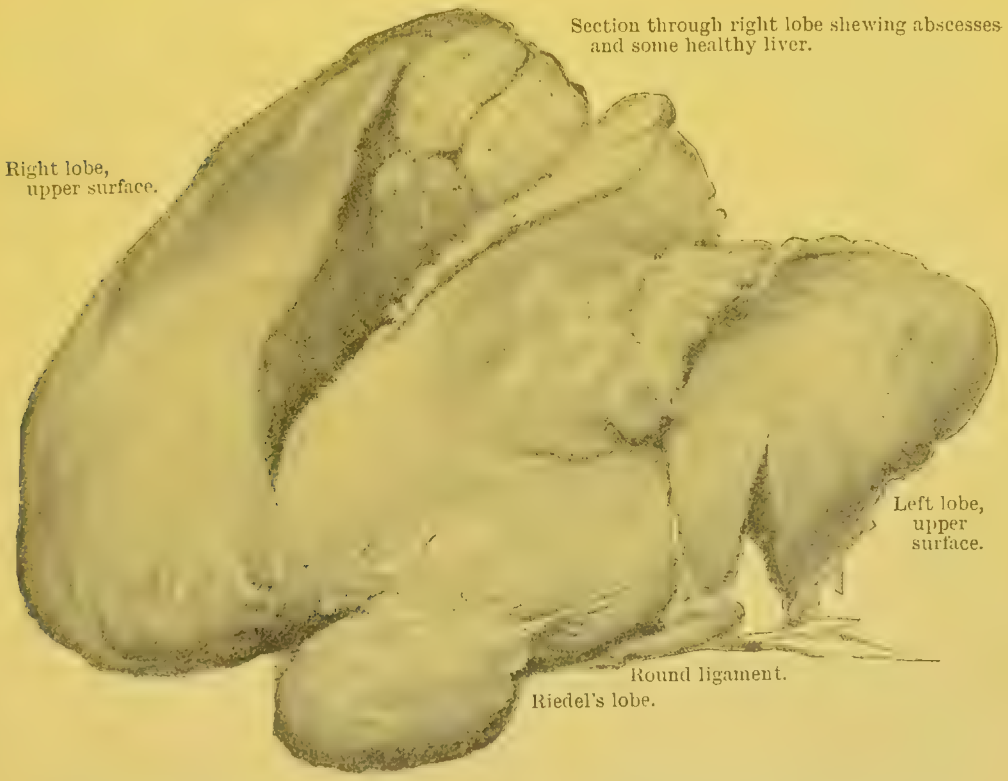
Section through right lobe shewing abscesses and some healthy liver.
Right lobe, upper surface.
Left lobe, upper surface.
Round ligament.
Riedel's lobe.

FIG. 8.—The liver with Riedel's tongue-like lobe and areas of suppuration due to suppurative cholangitis. From a case of cholecysto-colic fistula due to gall-stones. (Drawn by Dr. E. A. Wilson.)

Incidence.—Tongue-shaped lobes are much more frequent in women. This depends on their causation, both tight lacing and cholelithiasis being much commoner in that sex.

Anatomy.—The tongue-shaped lobes may either taper off gradually into an elongated, thick process from the right lobe, or the connecting pedicle may be reduced to two layers of somewhat thickened peritoneum. In the latter case the tongue-like lobe is freely movable, and during life may appear to be quite distinct from the liver. The gall-bladder may, but need not, be situated on the under surface of the tongue-like lobe, as in two cases excised during life (Martin, Bastianelli); the cystic duct will then run across the pedicle. As a rule, the tongue-like lobe is not perfectly normal in structure; from repeated attacks of congestion it may shew fibrosis, and degeneration, atrophy, and pigmentation of the liver cells, and haemorrhages. It has been found to be gummatous when the remainder of the liver was healthy, and may be the seat of secondary new-growth (compare p. 9).

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[1] McPhedran. *Canadian Practitioner*, 1896, xxi, 401.

[2] Keith, A. "The Anatomy of Glénard's Disease," *London Hosp. Gaz.*, 1902, ix, 55.

Roux[1] described primary carcinoma starting in a tongue-like lobe. In his case there was calculous cholecystitis. In similar cases care must be taken to see that the growth does not start in what is a much commoner situation, viz. the walls of the gall-bladder.

Physical Signs.—There is a movable abdominal tumour on the right side of the abdomen, which descends with the diaphragm and the liver on respiration. It is dull on percussion, but this is seldom continuous with the liver dulness, as there is usually a band of resonance between it and the liver. It may be distinctly tender on pressure. It may descend as low as the right iliac region and give rise to difficulty in diagnosis from appendicitis, etc.

Symptoms.—The existence of a tongue-like lobe may be discovered accidentally and may not be accompanied by any bad effects. Usually the symptoms associated with the existence of a tongue-like lobe are due to calculous cholecystitis, which may have played an important part in the production of the deformity. The patient may have been conscious for some time of enlargement and alteration in shape of the abdomen and of a slowly growing tumour, as in Martin's case in which a tumour had existed for twelve years. There are often a feeling of oppression and heaviness in the right hypochondrium and pain in the back. Abdominal pain may be paroxysmal, like gall-stone colic, or may be constant; the pain is usually relieved by rest in the recumbent position. Intense venous engorgement of the lobe may give rise to attacks of palpitation, vomiting, and collapse. Jaundice and ascites are rare and are due to some definite cause, such as gall-stones or chronic peritonitis.

Diagnosis.—Riedel's lobe may appear to have no definite connexion with the liver, and thus appears as a movable abdominal tumour and may easily be mistaken for a floating kidney, as in C. Martin's case.[2]

A woman aged thirty-six had had a lump in her abdomen for twelve years which had recently grown rapidly and become tender and painful. An oval tumour about the size of a six months' pregnancy filled the right half of the

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[1] Roux. *Rev. méd. de la Suisse Rom.*, 1897, xvii, 114.
[2] Martin, C. *Birmingham Med. Rev.*, 1898, xliii, 92.

abdomen ; it was tense and very mobile, dull on percussion, and separated from the liver by a band of resonance. It was thought to be an unusually mobile kidney. Laparotomy revealed a pedunculated accessory lobe of the liver bearing the gall-bladder ; it was successfully removed. Its weight was 3¾ pounds.

Bastianelli [1] described a case in which a displaced cancerous kidney was diagnosed ; laparotomy revealed a floating lobe of liver, with the gall-bladder on its under surface. This piece, weighing 500 grams (18·75 ounces), was successfully removed ; there were gummas in it, but not in the remainder of the liver.

A tongue-like lobe may be thought to be some other form of abdominal tumour, such as a solid growth in the omentum, a pyloric tumour, a distended gall-bladder, or a cyst of the pancreas or mesentery, or in extreme cases a uterine fibromyoma, ovarian cyst, or appendicitis.

In a case under my care in St. George's Hospital the gall-bladder, greatly thickened from calculous cholecystitis, was adherent to the colon, and was associated with a well-marked tongue-like lobe which before laparotomy was thought by some to be a floating kidney or a growth in the transverse colon. A Riedel's lobe containing two gummas was in one case diagnosed as carcinoma of the caecum (Munro [2]).

Treatment.—In cases in which a tongue-like lobe is diagnosed, treatment is only necessary when pain or discomfort is present. If there is any underlying cholecystitis or cholelithiasis, treatment should be directed to those conditions. In seven cases in which the morbid condition of the gall-bladder was treated, the tongue-like lobe disappeared (Terrier and Auvray [3]). An ill-fitting corset or one which presses on the liver should be replaced by a straight-fronted corset, and tight lacing or constriction of the waist by a belt should be prevented. It is not often that radical treatment, such as stitching the lobe to the abdominal parietes or its complete removal, is necessary.

In three cases the tongue-like lobe has been stitched to the abdominal parietes with success (Billroth,[4] Tscherning,[5] Langenbuch [6]). Removal of the lobe has been carried out by Bastianelli, Martin, and Lockwood.[7]

Effect of Tight Lacing on the Gall-bladder.—The gall-bladder is frequently dilated. Hertz [8] found it so in 24 out of 41 cases. The downward displacement of the duodenum brings tension to bear on the cystic duct, which even under normal conditions requires a spiral valve

[1] Bastianelli. *Policlinico*, April, 1895 ; *Brit. Med. Journ.*, 1895, i, epitome, No. 337.
[2] Munro. *New York State Journ. Med.*, 1908, viii, 183.
[3] Terrier et Auvray. *Rev. de chir.*, 1897, xvii, 735.
[4] Billroth. *Wien. med. Wchnschr.*, 1886, No. 14.
[5] Tscherning. *Centralbl. f. Chir.*, 1888, xv, 426.
[6] Langenbuch. *Ann. Soc. méd.-chir. de Liége*, 1888, xxvii, 414.
[7] Lockwood, C. B. *Lancet*, 1903, ii, 223.
[8] Hertz. *Abnormitäten in der Lage und Form der Bauchorgane*, 1894.

to keep it open (Keith[1]), and thus leads to obstruction. The resulting retention of bile and mucus in the gall-bladder may tend to elongation of the right lobe, and so give rise to, or accentuate, the tongue-like lobe seen in many corset livers. Retention of bile in the gall-bladder disposes to cholecystitis and cholelithiasis, and the predominance of female sufferers from gall-stones is no doubt partly due to the effects of the corset.

When calculi are present in the gall-bladder, the pressure exerted by the corset may, as suggested by Fütterer,[2] increase the friction between the gall-stones and the mucous membrane of the gall-bladder, and thus help to explain the greater frequency of primary carcinoma of the gall-bladder in women. In this connexion it is noteworthy that the larger bile-ducts, which are not affected in the same way by corsets or belts, are the site of primary carcinoma more often in males than in females.

DISPLACED LIVER

Apart from enlargement due to tumours or other causes, the liver may occupy an abnormal position under numerous and very various conditions. A displaced liver which is at the same time freely movable is a wandering liver (*vide* p. 22). Here we are only concerned with displaced livers which are not more movable than normal. Malposition may conveniently be divided in the first instance into (*a*) the congenital and (*b*) the acquired; the latter class is by far the larger.

CONGENITAL MALPOSITIONS

Transposition.—In complete transposition of the viscera the liver will be on the left side, but occasionally the heart may be congenitally transposed without the liver sharing in the change. Transposition of the liver without the other viscera being affected is very rare indeed.

Complete transposition of the viscera may lead to an erroneous diagnosis: in a patient with *situs transversus* cholelithiasis was diagnosed as acute yellow atrophy from absence of the hepatic dulness on the right side.[3]

Congenital Ectopia of the Liver (*Synonyms*: Hepatomphalos, Hepatocele).—From congenital defect of the muscles of the abdominal wall the liver may project under the skin either at the umbilicus or in the middle line between the umbilicus and the ensiform cartilage. Congenital hernia or ectopia of the organ at the umbilicus has been called hepatomphalos. The tumour is firm, dull on percussion unless coils of intestine are also present in the sac, and is continuous with the liver dulness. It may be easily reduced, but reappears on removal of the

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[1] Keith, A. *Lancet*, 1903, i, 639.

[2] Fütterer, G. Chicago Med. Soc., April 1, 1897.

[3] Billings. *Phila. Med. Journ.*, 1900, vi, 670.

Of Cullens 12 collected
cases 2 were not
in newly born

add x-ray

Cullen, T. S. Arch. Surg., Chicago, 1925, xi, 718 pressure. On the other hand, the liver may become adherent to the walls of the umbilical sac and thus be irreducible. This was the case in a baby aged three weeks.[1] Bullard[2] found the liver herniated into the umbilical cord.

Displacement of the Liver as the Result of Congenital Diaphragmatic Hernia.—Congenital defects of the diaphragm may give rise either to a free communication between the peritoneal and pleural cavities or, from deficiency of the muscular tissue only of the diaphragm, to a membranous pouch which projects up into the thorax. Congenital deficiency of the diaphragm is commoner on the left side (Jaffé[3]) and may allow the left lobe of the liver to enter freely into the thorax.

In a case of Porak and Durante's[4] the peritoneum communicated freely with the left pleura, and the left lobe of the liver was found behind the sternum.

ACQUIRED DISPLACEMENTS

In Diaphragmatic Hernia.—This may occur as part of a severe injury, the diaphragm being torn across, and the stomach, colon, spleen, or part of the liver may pass into the thoracic cavity. Diaphragmatic hernia when found in adult life without any history of injury may of course be congenital in origin, and in cases in which the herniated viscera have been found enclosed in a sac composed of the attenuated remains of the diaphragm or merely of the opposed pleura and peritoneum, this would appear probable. Pouching due to weakness near the oesophagus or xiphoid cartilage may, however, be acquired. In some cases a rent in the diaphragm may have been due to an injury received years before and almost or quite forgotten. The nature of the defect may then be regarded as congenital, whereas it is really acquired. In a considerable number of cases of diaphragmatic hernia the condition is found to have been preceded by trauma years before.

Traumatic diaphragmatic hernia is nearly always on the left side, since the support provided by the liver tends to protect the right leaflet from rupture. The liver is much less often displaced into the cavity of the thorax than the stomach, colon, intestines, or spleen.

In 300 cases the liver was herniated in 60 (Rochard[5]). In one case in which there was a history of a fall fourteen years before, part of a cancerous liver projected into the left pleura (Dietz[6]).

Part of the liver may pass through the rent and become tightly constricted at the margin of the rent like a strangulated hernia.

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[1] J. Hutchinson. *Med. Times*, 1870, i, 397.
[2] Bullard. *Am. Med.*, 1902, iv, 742.
[3] Jaffé. *Trans. Path. Soc.*, 1894, xlv, 224.
[4] Porak et Durante. *Bull. Soc. Anat.*, Paris, 1901, lxxvi, 354.
[5] Rochard. *Les Hernies*, p. 382, Paris, 1904.
[6] Quoted by Blum et Ombrédanne. *Arch. gén. de méd.*, 1896, i, 1, 178.

The following case, the necropsy of which I saw, was recorded by C. Ogle.[1] A boy, aged sixteen years, who had had an accident six years before, was admitted with frequent vomiting and signs of a left pleural effusion; aspiration did not remove any fluid. At the necropsy the cardiac end of the stomach and the left lobe of the liver were found in the left pleural cavity, having passed through a rent in the central tendon of the diaphragm, which had a diameter of 1½ inches. The intrathoracic part of the left lobe was connected by a thin atrophied pedicle to the rest of the liver and formed a lump 5 by 4 inches which looked very like a hard, deeply congested spleen. On section it was deep red in parts with white areas, and was mottled. Microscopically there were much haemorrhage, a quantity of blood-pigment, and degeneration of the liver cells.

In Spinal Curvature and in Rickets.—The liver though structurally normal may be very considerably displaced by spinal deformity. Its lower edge may be below the umbilicus and the organ may be unduly movable. In rickets the deformity of the chest to some extent depresses the liver, which is usually somewhat enlarged from the action of intestinal poisons. The view that the liver is enlarged in rickets is true, but the increase in the downward extent of the hepatic dulness is often partly due to displacement by thoracic deformity.

Tight Lacing, etc.—The deformities of the liver resulting from tight lacing and artificial constriction of the lower part of the thorax are described elsewhere (p. 8). In these conditions the liver may be displaced upwards as a whole, or even downwards, according to the position of the "waist," but most commonly the right lobe is elongated downwards and has a constricted area, corresponding to the line of pressure, uniting the floating lobe to the main part of the right lobe.

Thoracic Conditions giving rise to Displacement of the Liver.—*Effusion into the Right Pleural Cavity.*—A large serous effusion or empyema will effect this, but inasmuch as these conditions are usually relieved by paracentesis, the best examples of displacement of the liver due to disease of the pleura are seen in cases of right pneumothorax. A neglected or latent pleural effusion may, however, reach a large size and then produce marked displacement of the liver.

A man aged forty-three years arrived at the hospital in a state of collapse and died in a few minutes. At the autopsy, which I performed, the right pleura contained 10 pints of pus, the liver was greatly depressed, its lower edge being on a level with the anterior superior spines of the ilia. A man sent up to me for a tumour of the liver had 8 pints of blood-stained fluid removed from the right pleura.

Besides being displaced downwards the liver is rotated on its antero-posterior axis. The right leaflet of the diaphragm being depressed or even presenting its convexity downwards, the right lobe is much more depressed than the left, and the left lobe swings upwards, the whole organ being pushed towards the left.

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[1] Ogle, C. *Trans. Path. Soc.*, Lond., 1897, xlviii, 114.

A pneumothorax on the right side may in rare cases lead to acute dislocation of the liver when the intrathoracic pressure is raised, as a result of a valvular communication between the lung and the pleural cavity.

Stiller[1] records a case in which a pneumothorax developed as the result of sneezing in a healthy man; the liver was depressed so as to form a large abdominal tumour.

A pleural effusion or pneumothorax on the left side may give rise, if excessive, to downward displacement of the left lobe of the liver.

Malignant Disease of Lung.—Very extensive malignant disease affecting the lung and greatly increasing its volume may displace the liver downwards.

This displacement was extreme in a boy with an enormous calcifying sarcoma in the thorax, secondary to sarcoma of the thigh, who died in St. George's Hospital in 1898. The growth pressed the right leaflet of the diaphragm down so that its under surface was convex, the liver was depressed, and its lower border was on a level with the umbilicus. It was not more freely movable than normal.

In mediastinal growths the liver is not displaced unless, as not infrequently occurs, there is a large pleural effusion.

In *emphysema* the downward displacement of the liver is often easily detected, but the increased downward extent of the liver is not extreme unless there is, in addition, chronic venous engorgement due to failure of the right side of the heart.

Pneumonia.—Bright[2] thought that pneumonia was the direct physical cause of the liver being below the costal arch. But the cloudy swelling of the liver is in part responsible for the projection of the liver downwards. When the whole lung is solid, the diaphragm may be in the position of maximum inspiration, and as a result the liver is somewhat depressed. I have seen downward displacement of the right half of the diaphragm and of the liver at the necropsy of a child with extensive tuberculous pneumonia of the right lung.

Pericardial Effusion.—A large pericardial effusion will depress the diaphragm and with it the liver, usually the left lobe.

In a case figured by Sibson,[3] in which the pericardium contained $3\frac{1}{4}$ pounds of fluid, the right lobe of the liver was displaced and tilted just in the same manner as in a right-sided pleural effusion.

Abdominal Conditions leading to Displacement of the Liver.—Generally speaking, abdominal conditions which displace the liver do so in an upward direction, but occasionally the liver may be depressed, while in other cases it may be pushed, drawn, or rotated laterally. Growths arising from the retroperitoneal space may push the liver forwards, and, conversely, gas free in the peritoneal cavity or under certain conditions tympanitic distension of the intestines may separate the liver from the anterior abdominal wall.

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[1] Stiller. *Wien. med. Wchnschr.*, 1901, xli.

[2] Bright, R. *Abdominal Tumours*, p. 255, 1860. New Sydenham Soc.

[3] Sibson. Russell Reynolds' *System of Med.*, 1877, iv, 307.

Abdominal Conditions displacing the Liver Downwards.—A subphrenic abscess on the right side may pass between the diaphragm and the convexity of the right lobe and displace the liver downwards. A right-sided subphrenic abscess may be the result of a gastric ulcer situated near the pylorus, of a perforating duodenal ulcer, though the resulting peritonitis unfortunately is rarely localised, and may then k/ contain gas as well as pus, or trac\ up from perforation of an inflamed appendix, situated in what is not an infrequent abnormal position, viz. running up over the right kidney. Besides these extrinsic origins for a right-sided subphrenic abscess, an abscess in the liver itself, a suppurating hydatid cyst, or the multiple abscesses of pylephlebitis may perforate into the potential space between the liver and diaphragm and give rise to a collection of pus there and so to downward displacement of the liver. In these cases the liver is itself enlarged and the condition is almost the same as hepatic abscess.

A hydatid cyst projecting from the convexity of the liver rather encroaches on the thorax than pushes the liver downwards. Shattuck [1] described a single cystic adenoma of the bile-ducts containing a gallon of clear fluid which pushed the liver downwards (*vide* p. 458).

As the result of peritoneal adhesions due to local peritonitis, *e.g.* from appendicitis or inflamed tuberculous glands (Treves [2]), the liver may be pulled downwards towards the pelvis. In large inguinal or femoral herniae the liver may be drawn down. A liver which in the first instance was freely movable may subsequently become fixed in an abnormal position by local peritonitis or perihepatitis; thus Richelot [3] found the liver fixed in the right iliac fossa close to the caecum.

Upward Displacement of the Liver.—Ascites, flatulent distension of the intestines, excessive dilatation of the colon, or the presence of large abdominal tumours, such as ovarian cysts or uterine fibromyomas, push the liver and diaphragm up and thereby encroach very seriously on the capacity of the thorax. The convexity of the diaphragm may then be on a level with the third rib or even higher.

As an example of extreme displacement of the liver from the pressure exerted by ovarian cysts reference may be made to a remarkable case recorded by F. A. Baldwin [4] in which there were four ovarian cysts, two large and two small, in the abdomen of a woman aged fifty-nine years. The cysts contained 18 gallons of fluid and were estimated to weigh 185½ pounds. The highest point of the liver was on a level with the upper border of the third rib in the nipple line.

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[1] Shattuck. *Boston Med. and Surg. Journ.*, 1900, cxlii 427.
[2] Treves, F. *Brit. Med. Journ.*, 1896, i, 1.
[3] Richelot. *France méd.*, Paris, 1893, xl. 449.
[4] Baldwin. *Brit. Med. Journ.*, 1900, ii, 80.

When this upward displacement is very considerable, the liver may largely or even entirely cease to be in contact with the anterior abdominal wall and undergoes a very striking alteration in its relation to other organs. The anterior surface travels backwards and becomes posterior, while the inferior surface looks forwards and upwards instead of downwards and backwards. This is due to the liver moving upwards on a transverse axis running through its connexion with the inferior vena cava, which is relatively a fixed point.

Displacement Forwards.—A retroperitoneal growth or a pancreatic cyst may in rare cases push the liver forwards, a growth of the right suprarenal capsule may displace the right lobe forwards, while a pancreatic cyst will tend to force the left lobe into undue prominence. An aortic aneurysm near the pillars of the diaphragm may so displace the liver forwards as to suggest a new-growth in that organ. In Beatty's [1] case of aortic aneurysm in this situation the liver appeared steadily to increase in size.

Displacement Backwards.—Occasionally coils of intestine or the colon may intervene between the liver and the anterior abdominal wall, thus displacing the liver backwards. This occurs in acute yellow atrophy in which the great diminution or even complete disappearance of the liver dulness is largely due to the flabby liver allowing resonant bowel to come between it and the abdominal parietes. In cases of perforation free gas in the peritoneum may pass between the liver and the anterior abdominal wall and displace the organ backwards. In rare cases a distended piece of small intestine or colon may get between the anterior surface of the liver and the abdominal wall. This accounts in all probability for the occasional absence of liver dulness seen in cases during life for which no definite cause, such as acute yellow atrophy, a subphrenic pyopneumothorax, or perforation, is forthcoming. In these cases, in which there may be no abdominal symptoms, a thickening of the capsule of the liver corresponding to the abnormally situated piece of intestine may sometimes be found after death. Dilatation of the stomach or extreme degrees of dilatation of the descending colon will tend to rotate the liver towards the right.

T. Fisher [2] figures cases of this kind and I have seen several examples of this condition. In one case the colon lay just anterior to the atrophied bridge of liver substance connecting the constriction lobe with the remainder of a tight-laced liver.

A displaced liver is, as a rule, not more movable than one in its normal position. It differs from a wandering liver in this respect, and also in the fact that it cannot be replaced in its normal position, while, in addition, a definite cause for its displacement is often forthcoming. Symptoms which might be referred to a displaced liver, such as weight, pain, and heaviness, are generally thrown into the shade by those of the condition responsible for the displacement. The various forms of enlargement of the liver, fatty, lardaceous, leukaemia, new-growth, abscess, cirrhosis, etc., must be differentiated from a displaced liver by a careful physical examination of each individual case.

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[1] Beatty. *Dublin Hosp. Rep.*, 1830, v, 166.

[2] Fisher, T. *Bristol Med.-Chir. Journ.*, 1901, xix, 215.

The **treatment** of a displaced liver is that of the causal condition.

HEPATOPTOSIS

Synonyms: Wandering Liver, Movable Liver, Prolapse or Dislocation of the Liver.

By the term wandering or movable liver is meant one which, being unduly displaceable, leaves its normal position and forms an abdominal tumour.

Historical.—Cantani[1] in 1866 described a clinical case of movable liver, but there was no necropsy. Though Cantani's name is connected with the recognition of its clinical features, the anatomical condition had been described long before.

Heister, as far back as 1754, published the account of an autopsy, with a plate shewing the position of the liver. Gunzius (1744), Buchoby (1768), and Sauvages (1768) also referred to cases. Wickham Legg[2] first drew attention to the subject in this country. In recent times Glénard[3] has done much to direct attention to the subject, and introduced the name hepatoptosis in 1892.

Hepatoptosis is analogous to a wandering spleen. Both these organs are normally "floating" in the abdominal cavity, for while tethered in their normal positions by peritoneal ligaments, they are supported by the mutual pressure of the other abdominal viscera, especially the elastic pad formed by the intestines, and are not fixed and packed round by fat in the way that the kidneys are. The term "floating," which is accurately applied to an unduly movable kidney, is an equally appropriate epithet for a normal liver or spleen, and therefore does not describe an abnormally mobile condition of these viscera. The liver normally moves during respiration, descending with the diaphragm on inspiration half an inch below the costal arch in the right nipple line. A dilated stomach or distension of the colon on the left side will rotate the liver to the right. When this displacement is greatly exaggerated, the state of affairs in wandering liver is imitated.

There is a great difference between an unduly movable liver on the one hand, and one which is merely pushed out of place by a tumour or pleural effusion on the other hand. A displaced liver is not necessarily, or indeed usually, more movable than one in the normal position. Again, the tongue-like and constriction lobes attached to the right lobe of the

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[1] Cantani. *Ann. univ. di med.*, Milano, 1866, clxxxviii, 373.

[2] Legg, W. *St. Barth. Hosp. Rep.*, 1877, xiii, 141.

[3] Glénard. *Les Ptoses viscérales*, Paris, 1899.

liver in tight lacing and other conditions must be distinguished from a wandering liver. The wandering liver may be spoken of as total hepatoptosis, while the constriction and tongue-like lobes have been called partial hepatoptosis.

Existence of Total Hepatoptosis.—From the collected cases of Faure, Einhorn, Graham, Bötticher, Glénard, and Telaky, it has been estimated by Dutton Steele[1] that, in all, about 100 cases of undoubted total hepatoptosis are on record, of which 44 have been confirmed by operation or autopsy. Its existence, therefore, admits of no reasonable doubt, but from its intimate attachment to the diaphragm, the liver is not so often affected by visceroptosis as the other abdominal organs.

The existence of movable livers has been doubted from time to time, and the clinical signs of the condition have been explained as due to causes such as floating kidneys, renal or other tumours. This was the view taken by Wickham Legg.[2] In some cases in which a floating liver was diagnosed during life the liver has been found in its natural position after death (P. Müller,[3] Crawfurd). The explanation of this is that after death gaseous distension of the intestines pushes the liver up and the diaphragm ascends.

According to Hertz[4] and Glénard,[5] some at any rate of the cases described as wandering livers were in reality constriction lobes attached to a thinned and elongated right lobe. The part below the corset furrow may thus be mistaken for the whole liver, especially when it is very mobile and the abdomen is lax and pendulous. Glénard, indeed, considers that the predominance of the female sex in the recorded cases of movable liver is thus explained.

Frequency.—Well-marked examples of hepatoptosis with definite symptoms are rare. Minor degrees, however, are fairly often found when looked for; thus Glénard, from researches on a large number (3500) of invalids suffering from various disturbances of nutrition, estimated that 20 per cent of these patients present some degree of it. He put the numbers at 25 per cent for males and 15 per cent for females, thus reversing the usual incidence of really movable livers in the two sexes. For there can be no doubt that cases with characteristic symptoms are usually met with in multiparous women with pendulous abdominal walls. It is noteworthy that Glénard has found some degree of hepatoptosis with greater frequency in his later papers; thus, in 1886, he estimated that it occurred in 2 per cent and in 1892 in 20 per cent of patients with diseases of nutrition. It is fairly safe to assume that many of the latter cases, which would escape observation in less practised hands, do not suffer from the effects of the condition.

Sex.—The female sex provides the vast majority of cases of wandering livers. In 117 cases of hepatoptosis collected from literature by Ssaweljew,[6] 103 were in women, 13 in men, and 1 in a child. In

[1] Dutton Steele. *Univ. Penn. Med. Bull.*, Phila., 1903, xv, 424.
[2] Legg. *St. Barth. Hosp. Rep.*, 1877, xiii, 141.
[3] Müller, P. *Deutsch. Arch. f. klin. Med.*, 1874, xiv, 146.
[4] Hertz. *Abnormitäten in der Lage und Form der Bauchorgane*, 1894.
[5] Glénard. *Les Ptoses viscérales*, Paris, 1899.
[6] Ssaweljew. *Arch. f. klin. Chir.*, Berlin, 1903, lxx, 644.

Graham's[1] 70 cases 56 were in women. Max Einhorn[2] in private practice noted 21 women and 9 men. As already mentioned, Glénard's own observations, which include minor degrees of hepatoptosis, were to the effect that hepatoptosis is really more frequent in men, its apparently greater incidence in females being explained by cases of partial hepatoptosis (Riedel's lobes, constriction lobes) erroneously regarded as complete hepatoptosis.

Age.—The majority of patients with hepatoptosis are over forty years of age. Treves[3] puts the age at between thirty-five and sixty. In exceptional instances it is met with in children.

Freeman,[4] in 496 autopsies on children, met with four examples of hepatoptosis due to relaxation and elongation of the suspensory ligament; the right lobe came down into the pelvis, the left lobe being the only part of the liver touching the diaphragm, while the upper surface was turned to the right.

Factors responsible for the Normal Position of the Liver.—As bearing on the causation of hepatoptosis it will be well to refer to the factors which maintain the liver in its normal position. These are: (1) The healthy tone of the abdominal muscles which keeps up the intra-abdominal pressure and makes the intestines act as an elastic pad or support for the liver. In the rare cases of congenital absence of the muscles of the abdominal wall the liver has been extremely movable (Guthrie[5]). (2) The attachment of the liver by the inferior vena cava to the diaphragm. This, according to Faure,[6] is the most important agent in retaining the liver in its place, and by itself is capable of sustaining weight of 27 kilograms, as against 20 kilograms which the other suspensory ligaments can support. (3) The peritoneal ligaments, the falciform, coronary, and lateral, and the connective tissue uniting the right lobe of the liver to the diaphragm. Graham's[7] observations on the dead body shewed that they can support the liver independently of the abdominal walls, and both he and Dutton Steele[8] found that cutting these ligaments led to a condition which was much the same as that in hepatoptosis. These observations shew that in spite of Symington's[9] statement that under ordinary conditions the suspensory ligaments are not tense, they can, when intact and healthy, prevent any abnormal or excessive descent of the liver.

Intrahepatic blood-pressure may possibly play a part in keeping the liver in its normal position. Injection of water into the vessels of the liver was found by Glénard and Siraud[10] not only to increase its size, but to straighten out its

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[1] J. E. Graham. *Canadian Practitioner*, 1895, xx, 399.
[2] Max Einhorn. *Med. Rec.*, N.Y., 1899, lvi, 397.
[3] Treves, F. *Lancet*, 1900, i, 1339. [4] Freeman. *Arch. Pediat.*, 1900, xvii, 81.
[5] Guthrie, L. G. *Trans. Path. Soc.*, 1896, xlvii, 139.
[6] Faure. *Thèse de Paris*, 1892.
[7] Graham, J. E. In Loomis and Thompson's *System of Practical Med.*, iii, 414.
[8] Dutton Steele. *Univ. Penn. Med. Bull.*, 1903, xv, 424.
[9] Symington. *Trans. Med.-Chir. Soc.*, Edin., 1887, vii, 53.
[10] Glénard et Siraud. *Lyon méd.*, 1895, lxxix, 171.

under or concave surface, which became more convex. It is further stated that in chronic engorgement of the liver the organ keeps well in its place even though the abdominal walls are flaccid. Alteration in the intrahepatic blood-pressure may therefore possibly have something to do with dropping of the liver.

Causation of Hepatoptosis.—Undue mobility of the liver might depend on some congenital defect or abnormality of the suspensory apparatus, such as an imperfect development of the falciform or coronary ligaments, or upon elongation, which when extreme might justify the term "mesohepar." There is very little anatomical evidence to support this hypothesis put forward by Meissner,[1] Clarke and Dolley's [2] case of mesohepar being most exceptional. It is quite reasonable to suppose that elongation of the suspensory ligaments may be congenital in the sense that there is an hereditary weakness which, like that underlying hernia and general dropping of the viscera (Glénard's disease), allows elongation of the ligaments to occur later in life under conditions which in ordinary persons would not have this effect. Another equally tenable view is that the tendency to elongation is acquired and depends on degeneration or atrophy of the suspensory ligaments induced by malnutrition and general debility. The associated enteroptosis, pendulous abdominal walls, and lineae albicantes may be regarded as manifestations of the same process, and, as will be seen later, they favour the production of hepatoptosis. Elongation of the suspensory ligaments must occur in order to allow the liver to become movable.

In cases in which the suspensory ligaments are unduly extensible, dragging on the liver by peritoneal adhesions, tumours, cysts, or large accumulations in the gall-bladder may lead to elongation of the ligaments, and so to an excessively movable and displaced liver. This condition resembles a simple displaced liver (*vide* p. 16), but differs from it in excessive mobility. Total hepatoptosis due to traction is a rare event, and must be distinguished from the elongation of the right lobe (Riedel's lobe, partial hepatoptosis) seen in many cases of cholelithiasis (*vide* p. 12).

Thus, in a case of Treves,[3] a young woman aged twenty-two years, the liver descended 2 inches on assuming the erect posture; there was general visceroptosis due to traction exerted by the great omentum, which was firmly adherent to calcified tuberculous glands in the right iliac fossa.

Tight lacing and corsets are probably extremely important in the production of a freely movable liver. The pressure of the corset and the traction exerted by a heavy skirt at the waist tend to displace the abdominal viscera downwards. The suspensory ligaments of the liver, if degenerate, would thus be easily elongated. Further, tight lacing leads to weakness of the abdominal muscles, and so increases the conditions favourable to hepatoptosis.

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[1] Meissner. Schmidt's *Jahrb.*, 1869, cxli, 107.
[2] Clarke, T. W. and Dolley. *Am. Journ. Med. Sc.*, Phila., 1905, cxxx, 969.
[3] Treves. *Brit. Med. Journ.*, 1896, i, 1.

Landau[1] and Hertz, however, who have paid considerable attention to the effects of tight lacing, oppose this view.

Failure of the healthy tone of the abdominal muscles, leading to a pendulous condition of the abdomen with a diminution of intra-abdominal pressure, is an important factor in hepatoptosis. A flaccid abdominal wall removes the support provided to the liver by the intestines, and thus throws the weight of the liver on to the suspensory ligaments, which if not sufficiently strong will stretch and elongate. According to Keith,[2] total hepatoptosis is always part of general visceroptosis. It has, however, been thought that weakness of the abdominal walls alone is not sufficient to cause hepatoptosis, and that, conversely, hepatoptosis may occur when the abdominal walls are healthy.

In 55 cases of hepatoptosis collected by Graham[3] only 19 were definitely stated to have pendulous abdomens.

The causes which lead to weakening of the abdominal parietes are chiefly those producing abdominal distension, such as repeated pregnancy, ascites, intra-abdominal tumours, persistent flatulence, and accumulations of fat. In addition, the wearing of stays and want of exercise diminish the healthy tone of the abdominal muscles, while anaemia, debilitating diseases, and neurasthenia have the same effect. This weakening of the abdominal walls is a most important factor in diminishing the intra-abdominal tension. As these conditions are more frequent in women, abdominal tension is much lower among them than among athletic men. In men who lead a sedentary life, such as tailors and cobblers, however, the intra-abdominal pressure may be low. In women it may even be negative (Bruce Clarke[4]).

Glénard[5] lays stress on "hepatism" as a factor in the production of movable liver. By "hepatism" he means a chronic nutritional change which may be hereditary or acquired, and may be of two kinds, (*a*) cholaemic, (*b*) uricaemic, corresponding to "arthritism" of some French authors, and to Murchison's "lithaemia." (*Vide* p. 39.)

To sum up: The important disposing factors in the production of hepatoptosis are (1) a weak, extensible condition of the suspensory ligaments, and (2) a low intra-abdominal tension due to atony of the abdominal walls. These conditions of impaired nutrition are very prone to go together and to be accompanied by general visceroptosis.

Associated Conditions.—*~~Viscer~~optosis.*—Hepatoptosis may be part of this general condition and its most prominent manifestation, or it may occur in cases in which there is no other manifestation of visceroptosis or only a floating kidney. The association of a floating right kidney with hepatoptosis is comparatively frequent.

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[1] Landau. *Die Wanderleber und der Hängebauch der Frauen*, Berlin, 1885.
[2] Keith, A. "Hepatoptosis," *System of Medicine* (Allbutt and Rolleston), iv, Part I. 18.
[3] Graham, J. E. Loomis and G. Thompson's *System of Practical Medicine*, iii. 414.
[4] Bruce Clarke, W. *Brit. Med. Journ.*, 1896, ii, 1493.
[5] Glénard. *Les Ptoses viscérales*, p. 736 *et passim*, Paris, 1899.

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In 44 cases of hepatoptosis, verified either by laparotomy or necropsy, Dutton Steele found 9 cases of floating right kidney, or 20·4 per cent. In 330 cases of nephroptosis recognised clinically by Glénard[1] in women, there were 70 cases of hepatoptosis.

Neurasthenia, etc.—As already mentioned, neurasthenic conditions and general debility with loss of muscular tone may accompany total hepatoptosis and are probably closely related to the low intra-abdominal pressure. Schwerdt,[2] indeed, regards visceroptosis as primarily dependent on muscular atony of nervous origin.

Exciting Causes.—A wandering liver in the majority of cases develops gradually. But in a certain proportion of the cases—according to Graham, in 5 per cent—there is an acute onset of symptoms suggesting sudden dislocation of the organ. This acute onset may in rare instances be due to severe injuries leading to rupture or laceration of the peritoneal suspensory ligaments, such as the passage of a wheel over the body, falls, or blows. In less extreme cases sudden exertion in lifting heavy weights, violent expiratory efforts, such as sneezing, coughing, vomiting, laughing, or straining, may have the same effect.

Max Einhorn[3] refers to a singer in whom the laborious work of the diaphragm incident to his profession probably led to laceration of the hepatic ligaments.

Forms of Total Hepatoptosis.—In hepatoptosis the liver tends to be rotated in two different directions: (1) On its transverse axis, so that its upper (diaphragmatic) surface comes in contact with the abdominal wall, while the anterior surface points downwards; this is anteversion. (2) On its vertical axis; usually the convexity of the liver is turned to the right and the under surface to the left.

The liver is more commonly displaced downwards and to the right, but it may be displaced downwards and rotated to the left. Thus, the liver may be (1) simply anteverted; (2) anteverted with rotation to the right, the usual form; (3) anteverted with rotation to the left.

Hepatoptosis with Anteversion.—The liver being more fixed posteriorly, where the inferior vena cava runs through it, than elsewhere, movement is least in this situation. The liver moves downwards, the sharp anterior margin sinking down towards the pelvis while the superior or diaphragmatic surface slides forwards and downwards so as to come under the anterior abdominal wall. The inferior surface of the liver at the same time becomes more posterior. The liver becomes much flattened out and elongated (*vide* Fig. 9). As the result of tight lacing, the front of the liver may shew a transverse line of fibrous atrophy. This grooved condition of the liver (*le foie cordé*; *Schnürleber*) when exaggerated has been spoken of as the "pilgrim's bottle liver" ("*le foie en gourde de pèlerin*").

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[1] Glénard. *Les Ptoses viscérales*, Paris, p. 503, 1899.
[2] Schwerdt. *Deutsch. med. Wchnschr.*, 1896, xxii, 53.
[3] Max Einhorn. *Med. Rec.*, N.Y., 1899, lvi, 397.

The following case, in which the condition was recognised only on the post-mortem table, is a good example of hepatoptosis with anteversion.

On opening the body of a widow aged fifty who died of bronchitis in St. George's Hospital, I found the liver extremely mobile, and occupying the front of the abdomen like a flattened cake and reaching 1½ inches below the umbilicus; both lobes were much elongated, as shewn in the figure. The coronary ligaments were very much elongated, measuring 2½ inches. There was also evidence of constriction from tight lacing; the lower part of the liver below the constriction could be easily turned up so as to form a double fold of liver substance. The commencement of the cystic duct contained a calculus, and the neck of the gall-bladder was much elongated. The kidneys appeared more movable than natural, and the presence of lineae albicantes on the abdomen as well as the character of the os uteri made it probable that she had had children. This case was evidently one of partially anteverted wandering liver. Somewhat similar cases are described by Crawfurd,[1] Peters,[2] and Griffiths.[3]

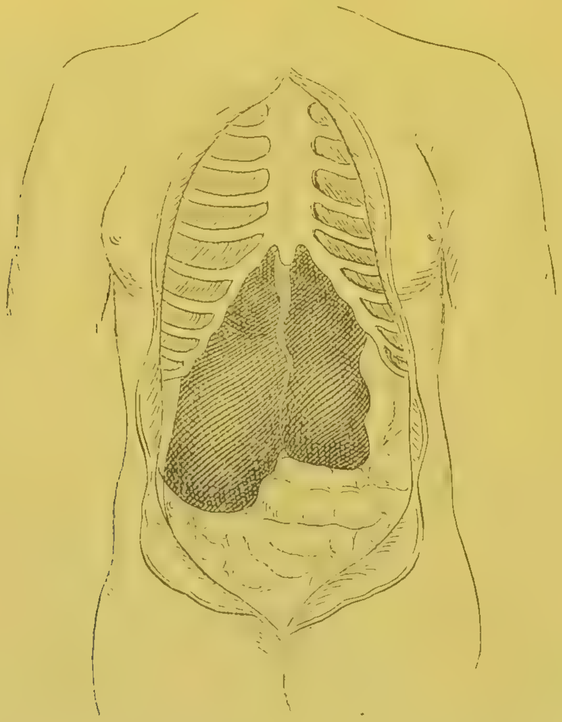

FIG. 9.—Partially anteverted liver with elongation of both lobes. (Drawn by Dr. H. B. Roderick.)

Hepatoptosis with Latero-version.—Rotation of the liver on its vertical axis takes place around the round ligament.

The following is a good example of a movable liver with rotation on its vertical axis to the right:—

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[1] Crawfurd, R. P. *Lancet*, 1897, ii, 1182.
[2] Peters. *Med. Gaz.*, N.Y., 1882, ix, 412.
[3] Griffiths, T. D. *Brit. Med. Journ.*, 1878, i, 89.

Morestin[1] found the gall-bladder and right lobe of a man's liver in the right iliac fossa. The liver was elongated and rotated so that the convexity pointed to the right and the under surface to the left. The left lobe was reduced in size to a mere tongue of hepatic tissue. There was no morbid change in the liver substance.

In cases of such rotation the right lobe may simulate disease around the appendix.

Complications.—Generally speaking, a movable liver can be replaced in its normal position, but this is not always possible; in 80 cases collected by Glénard this could not be effected in 14, while in 3 more it could only partially be accomplished. A movable liver may contract adhesions when displaced downwards, and so become fixed to the lower part of the abdomen. In these circumstances it readily gives rise to great difficulty in diagnosis and may resemble some abdominal tumour or inflammatory formation.

Richelot,[2] in an exploratory laparotomy for an abdominal tumour of doubtful origin, found the liver rotated and fixed in the right iliac region.

A movable and displaced liver has been found to be cirrhosed, to be associated with calculous cholecystitis,[3] gall-stones, or in exceptional instances to be occupied by malignant disease, gummas (Clarke and Dolley[4]), or a hydatid cyst (Nedwill[5]). A certain amount of atrophy and subsequent fibrous substitution may be due to torsion and twisting of the portal vessels and bile-duct in the lesser omentum.

In 80 cases collected by Glénard[6] the liver was healthy in 50; in 26 it was the subject of disease, usually cirrhosis or gall-stones. In one each cancer and hydatid cyst was present.

Physical Signs.—There is an abdominal tumour which is readily displaceable, and can be put back into the normal position of the liver, to which, indeed, it tends to return when the patient lies down, only to fall when a sitting or erect posture is assumed, the organ dropping two or more inches. The liver still descends on respiration; but the more marked the displacement, the less is this apparent. When the organ is very freely movable, and when presumably the lateral ligaments are greatly stretched or but ill developed, it can be readily rotated on its vertical axis, which passes through the inferior vena cava. This rotation is an exaggeration of that which a dilated stomach or colon may induce in the liver under normal conditions. The freedom of movement may, indeed, be so marked that the liver seems to turn over in its descent. When the patient turns on the left side, it travels in the same direction.

[1] Morestin. *Bull. Soc. anat.*, Paris, 1896, lxxi, 201.
[2] Richelot. *France méd.*, Paris, 1893, xl, 449.
[3] Lennander. *Gaz. d. hôp.*, Paris, 1900, lxxiii, 545.
[4] Clarke, T. W., and Dolley. *Am. Journ. Med. Sc.*, Phila., 1905, cxxx, 969.
[5] Nedwill. *Lancet*, 1901, ii, 914.
[6] Glénard. *Les Ptoses viscérales*, p. 625, Paris, 1899.

The displaced liver is visible under the relaxed abdominal walls as a rounded tumour on the right side about the level of the umbilicus. It is dull on percussion, firm, and smooth. The outline of the liver and perhaps the depression for the fundus of the gall-bladder and the notch between the left and right lobes for the round ligament can also be made out. When the organ has fallen away from the right hypochondrium, the normal liver dulness is replaced by resonance. In such cases the hand may be passed some way over the upper surface of the liver, between it and the diaphragm. There is a sinking or hollow in the right hypochondrium and a compensatory swelling or tumour, formed by the displaced liver, in the right flank or in the abdomen below the umbilicus. The empty state of the upper part of the abdomen below the right costal arch may be very striking.

The abdominal walls may be so lax and thin as to allow peristaltic action to be plainly visible, and divarication of the recti and a pendulous condition of the abdomen may be brought out when the patient rises from the horizontal position. According to Glénard, the lower part of the umbilicus becomes hidden by a fold of skin, upon which tension is brought to bear by the displaced liver through the attachment of the round and falciform ligaments. Other manifestations of visceroptosis, such as a floating kidney, a displaced uterus or stomach, may be present.

Onset.—Generally this is insidious and attracts no attention, but in some instances (Graham[1] says 5 per cent) it may be sudden, and then resembles a traumatic dislocation. There is then a feeling of something giving way, accompanied by sudden, twisting pain, which may be so severe as to make the patient faint, and may then be regarded as biliary colic.

A good case of sudden dislocation of the liver is recorded by Garnett[2] in a woman aged fifty, who, when hurriedly stooping to pick something up from the floor, felt a sudden wrench in the right side of the abdomen. The liver was found reaching to the right iliac crest. Rest in bed and the application of an appropriate bandage were followed by recovery. Graham[3] reported a movable displaced liver due to the wheel of a wagon passing over the abdomen in a boy aged seventeen.

Symptoms.—The symptoms of hepatoptosis may be summed up under different heads; cases may present the features of one or more of the following symptom-groups: (1) Pain, dragging, and heaviness in the hepatic region. (2) Symptoms referable to the intestinal tract, viz. dyspepsia, vomiting, and mucous colitis. (3) Imitating biliary colic, viz. pain and jaundice. (4) Imitating hepatic cirrhosis, viz. ascites, haematemesis, melaena. These symptoms are very rare. (5) Symptoms belonging to the respiratory system, viz. cough, dyspnoea. (6) Hypochondriasis, hysteria. There may, however, be no symptoms at all.

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[1] Graham, J. E. Loomis and Thompson's *System of Practical Med.*, iii, 415.
[2] Garnett, A. *Am. Journ. Med. Sc.*, 1881, lxxxi, 110.
[3] Graham, J. E. *Canadian Practitioner*, 1895, xx, 399.

(1) *Hepatic Pain.*—When symptoms are present, the commonest is a feeling of discomfort, weight, or actual pain in the right hypochondrium. The dragging feeling is commonly felt in the right hypochondrium or epigastrium, but it may be more extensive and radiate behind the sternum or even to the base of the neck, the traction exerted on the diaphragm by the liver being perhaps transmitted through the pericardium to the cervical fascia. Dull pain in the region of the right shoulder is common, and has been explained by traction on the diaphragm stimulating the phrenic nerve which communicates with the nerve to the subclavius muscle. The pain and discomfort are usually relieved in the horizontal position, but are aggravated by movement, and may quite prevent the patient from walking or getting about, or even from lying on the right side. The pain, however, may be constant and very distressing, and is then quite probably due to chronic cholecystitis, cholelithiasis, or stretching of adhesions. There are frequently attacks of very severe pain, exactly like biliary colic, but not necessarily accompanied by jaundice (*vide infra*).

In 44 cases of hepatoptosis these colicky attacks occurred in 37, or 84 per cent, and were accompanied by jaundice in 14 only (Dutton Steele [1]).

(2) *Symptoms referable to the Intestinal Tract.*—In other cases the symptoms are mainly dyspeptic. Nausea and vomiting may occur and may be set up by lying on the right side. Intestinal disturbance, such as flatulent distension and constipation, may be present. Glénard [2] considers that the vascular disturbance resulting from a wandering liver is the cause of mucous colitis, which he thinks is often associated with enteroptosis. Probably many symptoms observed in hepatoptosis, such as hysterical mental disturbance and irritability, leucorrhoea, menorrhagia, albuminuria, are rather the results of enteroptosis in general than of hepatoptosis in particular.

(3) *Symptoms imitating Biliary Colic.*—As already mentioned, attacks of pain resembling biliary colic are very frequent in wandering liver.

In 44 cases in which the liver was seen to be displaceable either at a laparotomy or necropsy, attacks of colic occurred in 37, or 84 per cent; in 14 of these 37 there was jaundice and in 10 calculi. In 15 cases attacks of colic and transient jaundice occurred without gall-stones (Dutton Steele).

The colic may be due to at least three causes, viz. calculi, the presence of a floating kidney on the right side,[3] or torsion of one of the bile-ducts. In the absence of gall-stones and a floating kidney on the right side the attacks of colic are probably due to torsion of the cystic duct at its junction with the common bile-duct, which would not necessarily cause jaundice or kinking of the common bile-duct at its commencement.

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[1] Dutton Steele. *Univ. Penn. Med. Bull.*, 1903, xv, 424.

[2] Glénard. *Académie de médecine*, April 20, 1897, 3 s, xxxvii, 465

[3] Compare J. Hutchinson, Jr. *Practitioner*, 1902, lxviii, 186.

In Crawfurd's[1] case of hepatoptosis with jaundice a twist seemed to have occurred at the commencement of the common bile-duct. In this case I had an opportunity of examining sections of the liver which shewed dilatation of the lymphatics and oedema in the portal spaces, as if the lymphatics had also been twisted and obstructed; but as bile had evidently passed into the general circulation, the obstruction must have been intermittent.

Dutton Steele, in discussing the question why jaundice is not constant, points out that the obstruction is not always in the common bile-duct and that the kinking may be very transitory, the liver returning towards its normal position. His experiments shewed that the further the liver was displaced towards the pelvis, the greater was the pressure required to drive an injection from the biliary papilla into the gall-bladder. A twist in the cystic duct may produce intermitting distension of the gall-bladder and attacks of biliary colic but without jaundice (Villard and Cotte[2]).

In some instances jaundice may occur without pain; Steele refers to two cases. It may be catarrhal (Snowman[3]); a displaced liver may by traction on the ducts lead to stagnation of bile in the ducts, and so favour hydrops of the gall-bladder, cholecystitis, and the production of gall-stones. In Newman's[4] case the gall-bladder contained 30 ounces of straw-coloured fluid and numerous gall-stones.

(4) *Symptoms imitating Hepatic Cirrhosis.*—In very exceptional instances, ascites, probably caused by twisting of the portal vein, and oedema of the legs, probably due to a kink of the inferior vena cava, occur. Twisting of the portal vein, by obstructing the circulation, might be expected to give rise to venous oozing into the stomach and intestines and so to haematemesis and melaena. Haematemesis is, however, extremely rare.

McNaughton Jones[5] described a case of a woman aged thirty-eight, the mother of seven children, who had haematemesis on several occasions, the first at the age of nineteen. The last attack was very severe, and at this time a tumour, thought to be renal, was discovered. At the laparotomy the tumour was found to be liver, which extended into the right iliac fossa.

(5) *Symptoms referable to the Respiratory System.*—In some cases, classed by Max Einhorn[6] as asthmatic, there is dyspnoea in addition to a feeling of fulness and constriction in the upper part of the abdomen.

A dry cough of eighteen years' duration, which, however, disappeared when the patient was in the recumbent posture, was found by L. Vène[7] to be associated with hepatoptosis, and was cured by the application of a flannel bandage. It was

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[1] Crawfurd. *Lancet*, 1897, ii, 1182.
[2] Villard et Cotte. *Rev. de chir.*, Paris, 1906, xxxiii, 331.
[3] Snowman. *Lancet*, 1896, i, 1281.
[4] Newman, D. *Brit. Med. Journ.*, 1902, ii, 249.
[5] McNaughton Jones. *Lancet*, 1898, i, 1327.
[6] Max Einhorn. *Med. Rec.*, N.Y., 1899, lvi, 397.
[7] Vène, Louis. *Journ. de méd. interne*, Paris, 1898, ii, 245.

thought that the cough was due to excitation of the diaphragm by traction of the liver on the inferior vena cava.

Some instances of cough usually put down to hysteria may in reality be due to hepatoptosis.

(6) *Hysteria* or *hypochondriasis* is frequently observed to be associated with hepatoptosis; the mental depression may result in the patient acquiring the morphia habit.

Absence of Symptoms.—A freely movable liver may be found accidentally in patients who do not suffer any inconvenience from it. Beddard[1] refers to a case in a placid unmarried lady who lived at her ease and in whom "the tumour" formed by the prolapsed liver merely aroused an "amused curiosity." It is probable that the presence or absence of symptoms largely depends on the life the individual leads and on her temperament. High-strung and nervous women often feel pain and suffer inconvenience from conditions, such as peritoneal adhesions and floating kidneys, of which their more robust sisters are unconscious.

Diagnosis.—The chief points in the diagnosis are the presence of a movable abdominal tumour, resembling the liver, which can be replaced in the position of that organ, and the fact that the hepatic region is sunken and resonant on percussion when the tumour is in the middle of the abdomen. In addition, other conditions, such as a floating kidney, must be excluded.

Differential Diagnosis.—A freely movable or wandering liver must be distinguished from an enlarged liver, from a tongue-like lobe with or without a distended gall-bladder, and from mere downward displacement of the liver such as may result from pleural effusion, pneumothorax, or other causes.

In Packard's[2] case of movable liver the diagnosis during life was subphrenic abscess following enteric fever.

From Floating Kidney.—The most frequent mistake seems to consist in regarding a wandering liver as a floating kidney or as some other form of enlargement of the right kidney. Floating kidneys are much commoner than hepatoptosis, and the general symptoms are so much alike that the condition of the liver may be overlooked, especially when, as is by no means infrequent, there is a floating kidney as well as a wandering liver. Alteration in the position of a movable liver may imitate an intermittent hydronephrosis; concomitant distension of the gall-bladder may have the same effect.

Newman[3] operated on a wandering liver and let out 30 ounces of bile from the gall-bladder. The symptoms had suggested a hydronephrosis.

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[1] Beddard, A. P. *Guy's Hosp. Rep.*, 1902, lvii, 179.
[2] Packard. *Trans. Coll. Phys.*, Phila., 1896, xviii, 230.
[3] Newman. *Brit. Med. Journ.*, 1902, ii, 249.

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A careful bimanual examination, if necessary under an anaesthetic, should be made; the liver dulness should be percussed, and an attempt to displace the abdominal tumour into the right loin should be made.

From Tumours and Hydatid Cysts in the Liver.—A wandering liver may suggest malignant disease of the organ, especially when, as in rare cases like Crawfurd's,[1] jaundice and ascites are present from torsion of the common bile-duct and portal vein in the lesser omentum. The ease with which the liver is displaced should prevent any mistake of this kind, while, on the other hand, irregularities of the surface of the liver and signs of chronic obstruction of the colon make malignant disease probable.

Hydatid cysts projecting from the upper surface of the liver, or, in rare instances, between the liver and diaphragm, give rise to downward displacement of the organ, but there is no absence of the hepatic dulness below the sixth rib in the right nipple line, as there would be if the liver had dropped away from its normal position. The converse mistake has occurred, and laparotomies undertaken for supposed hydatid cysts have led to the discovery of a wandering liver (Marchant,[2] Areilza[3]).

Gall-stones.—The pain and jaundice may, as in a case of Mathieu's,[4] lead to a diagnosis of biliary colic. Max Einhorn[5] has seen five cases of wandering liver that had previously been erroneously diagnosed as cholelithiasis. The two conditions may both be present.

Tumours and Inflammatory Thickenings of the Omenta, Mesentery, etc.—Tumours and cysts of the omentum and of the mesentery, though movable, cannot be displaced to the same extent from above downwards as a wandering liver; moreover, they are separated by a zone of resonance from the liver dulness. Inflammatory thickenings in the omentum being comparatively fixed, are hardly likely to cause any difficulty in diagnosis. In exceptional cases the question might arise whether a movable tumour is the liver or a mass of growth in the stomach, colon, or inflammation around the appendix; but under ordinary conditions there is little or no resemblance between hepatoptosis and these conditions.

Treatment.—Tight lacing and corsets which constrict the lower part of the thorax and tend to displace the liver downwards must be discontinued. The "straight-fronted" corset should be worn, as it supports the abdomen from below and does not compress the waist unduly. A suitable abdominal belt, binder, strapping, or elastic bandages should be applied so as to support the lower abdomen in an upward direction, and thus, by increasing the intra-abdominal pressure, to support the liver in its proper position. The belt or binder must reach below the hips and should be fixed in position while the patient is still in bed in the morning and before the liver has become prolapsed. This method of replacing

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[1] Crawfurd, R. *Lancet*, 1897, ii, 1182.
[2] Marchant, G. *Acad. de méd.*, Paris, Aug. 11, 1891.
[3] Areilza. *Rev. med. y cirug. práct.*, Madrid, 1896, xxxvii.
[4] Mathieu. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1893, 3. s., x, 639.
[5] Max Einhorn. *Med. Rec.*, N.Y., 1899, lvi, 397.

Prognosis is good as regards life. Apart from operative measures, the results of which are good, treatment is palliative only.

the healthy tone of the abdominal muscles is much more effective than applying a supporting pad to the liver alone. Rest in bed will, for the time being, relieve the pain, and it is advisable to raise the foot of the bed so that the hips are higher than the head. The treatment of movable liver is on the same lines as that of enteroptosis or Glénard's disease.

Diet is a matter of importance, and, as a rule, the patient requires liberal feeding so as to improve the general state of nutrition. The subjects of hepatoptosis are usually weakly, but when there is decided corpulence, the amount and quality of food taken should be supervised by the medical attendant, and any excessive eating interdicted. There will be no difficulty in distinguishing the weakly, flabby patient whose muscles and tissues are in want of better food from the heavy eater whose liver and tissues are congested from the presence of the products of excessive protein metabolism. It is important to keep the bowels freely open, for in this way portal congestion and flatulent distension are relieved and a distended gall-bladder may be emptied, conditions which may conceivably dispose to or exaggerate hepatoptosis.

In order to improve the tone of the abdominal muscles carefully planned gymnastic exercises may be employed. The exercises should be simple and readily carried out. Lea[1] speaks of the following as very efficient: (1) The patient lies on the back without any pillows, the arms being folded in front, and gradually raises herself into the sitting posture without any help from the arms. (2) The patient, being in the same position as before, raises first one lower limb, then both, with the knees extended, to a right angle with the trunk. (3) Deep breathing, especially inspiratory movements with the glottis closed and after a forced expiration. This exercise is useful in drawing up the viscera. Lea directs that each of these exercises should be performed six to twelve times night and morning.

Massage may be used in order to improve the muscular tone.

Electrical stimulation has been applied to the abdominal muscles and has been found to be beneficial. Griffith[2] obtained a good result by stimulating the muscles with an interrupted current daily for three months, but the long duration of the treatment would render it unsuitable in many cases.

If the application of a belt and the other palliative measures mentioned fail to relieve the symptoms, and the pain be so severe as to incapacitate the patient from ordinary life and work, the advisability of surgical interference must be considered. The operation of fixing the liver by sutures or other means in its proper position is known as hepatopexy or hepatorrhaphy. Various methods have been adopted, such as passing sutures through the liver substance and the abdominal wall, tying the round ligament up to the cartilage of the seventh rib and at the same time promoting adhesions between the diaphragm and convexity of the liver by rubbing the peritoneum with aseptic gauze. When the gall-

[1] Lea, A. W. W. *Med. Chronicle*, 1902, xxxvi, 225.

[2] Griffith. *Brit. Med. Journ.*, 1878, i, 89.

bladder contains calculi, the operation for cholecystotomy usually leads to fixation of the liver.

In 1891 Gérard Marchant[1] fixed the anterior margin of the liver to the costal margin by four silk sutures. Treves[2] utilised the round ligament to support the sutures. Lanelongue and Faquet[3] in 1895 sutured the liver to the anterior abdominal wall and roughened the opposed surfaces of the liver and diaphragm so as to get adhesions. Union of the liver to the opposed peritoneal surface of the diaphragm has also been obtained by swabbing the surface of the liver with strong carbolic acid. Péan[4] supported the liver by uniting the peritoneum of the anterior abdominal wall with that of the postero-lateral part of the abdomen below the replaced liver.

In 18 cases of hepatopexy collected by Terrier and Auvray,[5] there were 15 cures; and of 25 collected by Cernezzi,[6] 22 were stated to be cured.

The drawbacks to operative measures are—(1) that the underlying condition disposing to enteroptosis in general and hepatoptosis in particular is not removed; (2) that the incision through the abdominal walls is very prone to become the seat of hernial protrusion, inasmuch as their tone and nutrition are especially defective.

On the other hand, hepatopexy may succeed after all other mechanical and palliative measures have failed, and it does so by compensating for, though not removing, the disposing factors of hepatoptosis. The danger of hernial protrusion in the scar is probably greater in old women with permanently pendulous abdomens than in younger women who are temporarily in a low state of nutrition from several rapidly succeeding pregnancies. In any case an abdominal belt should be worn after the operation.

With regard to *prophylaxis*, tight lacing should be prevented and care should be taken that a pendulous condition of the abdominal wall is not induced or aggravated by getting up too soon after parturition and by neglecting the use of a proper binder.

FUNCTIONAL DISEASE OF THE LIVER

BEFORE entering on the rather difficult subject of functional disease of the liver it will be convenient to enumerate the important functions of the liver.

(1) The secretion of bile.

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[1] Marchant. *Acad. de méd.*, Paris, August 11, 1891, 3 ser., XXVI, 270.
[2] Treves. *Brit. Med. Journ.*, 1896, i.
[3] Lanelongue et Faquet. *Gaz. hebd. d. sc. méd. de Bordeaux*, 1895, xvi, 411.
[4] Péan. *Congrès de chir.*, Paris, 1896. Quoted by Treves, *Lancet*, 1900, i, 1344.
[5] Terrier et Auvray. *Chirurgie du foie et des voies biliaires*, 1901.
[6] Cernezzi. *Rif. med.*, 1910, xxv, 877.

The formation of urea is mainly the result of the conversion by the liver of amino-acids and ammonia absorbed from the [illegible] intestines into urea.

(2) The metabolic processes in connexion with the formation, storage, and hydrolysis of glycogen.

(3) In connexion with protein metabolism. The ~~liver is very largely concerned in the conversion of ammonia into urea~~, but there is no reason to believe that ~~it~~ is of the same importance in the production of uric acid. In some diseases, in which the liver cells are degenerated, the amount of urea is diminished while the quantity of nitrogen excreted in the urine as ammonia is increased. It has, therefore, naturally been assumed that the fall in the output of urea was due to failure in the functional activity of the liver cells. This, however, is not the ~~true~~ explanation. whole The real reason is that in these diseases organic acids, of the fatty acid series, are formed and unite with any available bases, among which is ammonia, present in the blood. The ammonia, which under ordinary conditions would be changed by the liver into urea, is now in a form which cannot undergo this transformation, and therefore appears in the urine linked with an organic acid. This occurs in acute yellow atrophy, phosphorus poisoning, and some cases of cirrhosis and fatty liver. In such cases ammonia given by the mouth increases the amount of urea in the urine, thus shewing that the liver cells have not lost their power of transforming ammonia into urea. It is only in the very latest stages of such diseases, shortly before death, that the liver cells appear to lose this power.[1]

(4) Its antitoxic or protective function. Poisons, whether introduced into the alimentary canal or manufactured there as the result of bacterial activity, are normally arrested, or converted into harmless bodies by the liver cells. The detoxicating action of the liver is shewn by the difference in the action of poisons, such as strychnine, conia, snake venom, albumoses, when introduced into the portal circulation, on the one hand, and into the general circulation, on the other hand. It appears from Roger's[2] observations that the antitoxic function of the liver varies with its richness in glycogen and its power of stopping sugar and forming glycogen, and that if the liver loses its power of stopping sugar it is also unable to arrest poisonous bodies brought to it from the portal area. In complete biliary obstruction the antitoxic power of the liver fails, and the grave manifestations seen under these conditions are chiefly due to the poisons which flood the body, and only depend in a minor degree on the presence of the bile in the blood.

From the number and importance of the functions of the liver it is clear that failure in discharge of these duties must be followed by very definite symptoms. Functional disturbance is undoubtedly common in the liver, but the following questions require some consideration:

(1) Are the disorders of hepatic function primary in the liver? and
(2) Are they entirely independent of structural change in the organ?

Numerous conditions, many of them in no way connected with the liver, were formerly described as due to functional disease of that organ. The idea was attractive to the lay mind and is recklessly appealed to in everyday life. Flatulence, dyspepsia, constipation, and the bad effects of overeating and drinking are often euphemistically described as "liver." As a reaction against this inaccurate though comforting doctrine most English medical writers at the present time ignore the subject or deny the existence of primary functional disease of the liver. This swing of the pendulum to the opposite extreme—for it must be admitted that the idea originated with the profession—is due to the knowledge that the symptoms ascribed to functional disease of the liver can in great part be explained as due to other factors, such as indigestion, auto-intoxication, constipation, or to subacute congestion or even inflammation of the organ, often secondary to intestinal disturbance or to absorption of toxic products from the intestine. In other words, the hepatic disturbances formerly regarded as due to primary functional insufficiency are in the vast majority of cases dependent on morbid processes elsewhere, and therefore secondary, or are associated with definite organic change in the liver.

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[1] For a lucid account of this question the reader should refer to Herter's *Lectures on Chemical Pathology*, p. 338, 1902, London.

[2] Roger. *Presse méd.*, Paris, 1897, i, 293.

Thus, to consider the symptoms commonly referred to functional disorder of the liver, the distaste for food, dyspepsia, flatulence, constipation or diarrhoea, are due to gastro-intestinal catarrh caused by poisonous or unsuitable food. The icteric tint of the conjunctivae, the muddy skin, and some of the mental depression are either due to the spread of the gastro-duodenal catarrh to the biliary papilla and the slight obstruction to the flow of bile thus induced, or possibly to catarrh of the minute intrahepatic ducts set up by poisons absorbed from the alimentary canal and subsequently excreted into the ducts; and in either case to the passage of bile, which often contains toxic constituents, into the general circulation. The pale colour of the faeces may be due to catarrh of the bile-ducts and a deficient amount of bile, but in cases without any jaundice elsewhere it has sometimes been regarded as due to deficient secretion of bile—acholia. Of this there is no proof; the colourless condition of the faeces may be due to several factors, increase of fat, a colourless form of urobilin, leuco-urobilin, due to over-reduction of urobilin, and often to permeation of the faeces with bubbles of gas from excessive carbohydrate fermentation. This colour of the faeces is often mentioned by the patients as proof that "the liver is not acting" and that there is an imperfect formation of bile. The increased amount of fat may depend on pancreatic rather than on hepatic disease, especially in children.

The headache, giddiness, muscae volitantes, malaise, muscular debility, mental depression, and irritability are due to the local action on the nervous system of poisons absorbed from the alimentary canal. These toxic bodies are either produced in such quantities that the liver fails to stop them, or more probably they act on the liver cells and impair their vitality and function; in either case the general circulation becomes flooded with toxic bodies. The piles, the feeling of weight in the right hypochondrium, and shoulder pain depend on hepatic congestion or

even slight hepatitis brought on by constipation and the advent to the liver of digestive products in excessive amounts and probably of altered (*i.e.* toxic) quality. This state of hepatic congestion is especially apt to be set up in patients who have suffered from malaria in the tropics. (*Vide* Tropical Liver.) Nevertheless there can be no doubt that in some instances morbid results are referred to the functional disturbance of the liver without its being always possible to determine satisfactorily that this disturbance is secondary. Thus, it is possible that in some forms of glycosuria there is an excessive activity of the glycogenic function, and that in alimentary glycosuria the liver fails to discharge efficiently its function of stopping the sugar brought to it by the portal vein. As a rule, in alimentary glycosuria there is some definite underlying cause, such as pancreatic disease, but it cannot be recognised in all cases. These conditions, however, are not ordinarily spoken of as functional disease of the liver.

It has recently been urged that puerperal eclampsia is in many cases due to hepatic insufficiency, and that the renal symptoms are secondary to a primary hepatic toxaemia. During pregnancy toxaemia is favoured by several factors, such as the retention of bodies which are normally removed in the menstrual flow, the passage of toxic bodies, derived from metabolic processes in the fetus, into the maternal circulation, and auto-intoxication from constipation, which is so common during pregnancy. In a woman who inherits a diminished hepatic activity and resistance the liver fails to rise to the occasion and to stop and destroy the poisons which reach it. The blood then becomes flooded with poisons, and symptoms of toxaemia result. These are dyspepsia, severe vomiting, ptyalism, oedema without albuminuria, pruritus, and pigmentation. In more marked instances there are intractable vomiting, jaundice, acute yellow atrophy, mania, and eclampsia. In fatal cases, however, changes are constant in the liver, and consist in focal or more extensive necroses, degeneration of the liver cells, and haemorrhages.

As has already been admitted, functional disorder of the liver is no doubt responsible for many symptoms. The difficulty in regard to the subject is to prove that the functional disturbance is primary in the liver and not secondary to disease or morbid factors elsewhere. The discussion is not a mere academic exercise, but has a practical bearing on the treatment. Thus, if it were thought that there was a primary failure of hepatic activity, as is implied by the common phrase "torpid liver," the rational course would be to stimulate the organ. Whereas, if there was an underlying and primary factor elsewhere, this should be attacked.

The difficulties about the recognition of primary functional disorders of the liver may be best explained by referring to some of the conditions which it has been supposed to cause.

Lithaemia was described by Murchison[1] as a condition of innate defect of power, often hereditary, in the liver, in virtue of which its healthy functions are liable to be deranged by the most ordinary articles of diet. As a result of this hepatic insufficiency uric acid, instead of urea, was said to be produced in the liver and turned out into the blood. Among the results of lithaemia Murchison enumerates such different conditions as dyspepsia, constipation, gout, urinary calculi, biliary calculi, and acute and chronic renal disease.

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[1] Murchison, C. *Croonian Lectures*, Royal College of Physicians, 1874; *Diseases of the Liver*, p. 594, ed. ii, 1877.

This conception has been revived in France under a different name—"hepatism"—by Glénard.[1] It is regarded as a chronic nutritional change which may be hereditary or acquired. It may take (*a*) the cholaemic form; this condition is much the same as the simple family cholaemia described by Gilbert and Lereboullet,[2] in which the blood-serum contains bile-pigment though the urine usually does not. Its primary signs are pigmentation of the skin, which may be of the nature of slight jaundice, or dark and resembling Addison's disease, moles, freckles, or brown areas (the biliary mask) resembling the melasma of pregnancy, and xanthoma of the eyelids. As secondary results such various conditions as the following may arise: dyspepsia, abdominal pain, haematemesis, epistaxis, mucous colitis, migraine, albuminuria, and rheumatic pains, while it is closely related to transient and chronic jaundice, biliary cirrhosis, and similar conditions. (*b*) The uricaemic form, which corresponds to the diathesis termed "arthritism" by some French writers, or to Murchison's lithaemia. Hepatism, according to Glénard, is a functional disturbance of the liver and is the cause of a large number of diseases, including those incriminated by Murchison, and, in addition, neurasthenia, diabetes, chlorosis, and visceroptosis. It may be pointed out that the cholaemic form probably depends on slight catarrh of some part of the bile-ducts, either in the liver or close to the lower end of the common bile-duct.

Murchison's conception of lithaemia is very far-reaching, and among other things offers an explanation of gout; in fact, many of the manifestations of lithaemia are those of irregular gout. Among recent writers Yeo[3] and Walker Hall[4] have supported the view that hepatic inadequacy is an underlying factor in the production of gout. The protean manifestations of neurasthenia include many of those formerly ascribed to functional failure of the liver.

This conception of lithaemia depends on the assumption that as the result of imperfect metabolism uric acid, instead of urea, is manufactured by the liver. There are two mistakes here. In the first place, it is now universally agreed that uric acid and urea are the products of entirely distinct metabolic processes, and that there is nothing to support the view that if the formation of urea is left incomplete uric acid results. As Woods Hutchinson[5] graphically expresses it, they are practically as distinct from each other as the urine from faeces. In the second place, it has been proved since Murchison's time that the liver does not play the predominant part in the manufacture of the uric acid. It is true

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[1] Glénard. *Les Ptoses viscérales*, Paris, 1899.

[2] Gilbert et Lereboullet. *Gaz. hebd. de méd.*, Paris, 1902, vii, 889.

[3] Yeo. *Brit. Med. Journ.*, 1901, i, 1457.

[4] Walker Hall. *Practitioner*, 1906, lxxvi, 366.

[5] Woods Hutchinson. *Lancet*, 1903, i, 288.

that the liver contains an oxidase which can transform xanthine and hypoxanthine into uric acid. But uric acid is produced in the body generally, and more especially from lymphoid tissue; it is closely associated with leucocytosis, uric acid being derived from the nuclein of the cells. This is well seen in the excessive excretion of uric acid in leukaemia. It is, therefore, too narrow a view of the faulty metabolism of protein material which results in an excessive production of uric acid to say that it depends on functional disorder of the liver to the exclusion of the rest of the body.

Habitual high blood-pressure and its accompaniments, such as migraine, might be thought to depend on a failure of the liver to stop and destroy the poisonous bodies that are continually being carried to it from the intestines. The liver undoubtedly exerts this important function of protecting the body from auto-intoxication, but it is difficult to prove that failure in the discharge of this duty leads to high blood-pressure; since in cases of extensive disorganisation of the liver—for example, in cirrhosis—hepatic insufficiency must exist, but the arterial tension is low and not raised. It is much more likely that high blood-pressure is, like gout, due to some general disorder of metabolism.

In cases popularly described as "biliousness" or "torpid liver" the symptoms are indigestion, some hepatic pain, headache, slight icteric tingeing of the conjunctivae with an apparent or real deficiency of colouring-matter in the faeces. The most probable explanation of these symptoms is gastro-duodenal catarrh with slight catarrhal jaundice and not a primary diminution in the secretion of bile. In these cases it is possible either that there is catarrhal swelling of the biliary papilla in the duodenum, or that, as the result of indigestion, poisonous products are carried to the liver and then, when excreted into the bile-ducts, set up a certain amount of catarrh in the small intrahepatic ducts. This leads to reabsorption of the bile with the poisons contained in it, which pass into the general circulation and act on the body as a whole. According to Sir Lauder Brunton,[1] the proverbial bitter taste of the bile is pathological and due to the presence of poisons absorbed from the bowel and then excreted into the ducts, healthy bile being tasteless.

It must be borne in mind that the liver, like other organs, must vary greatly in different individuals as to its functional activity and reserve power, and that an amount of food-products which can be satisfactorily dealt with by the liver in one individual would in another be entirely beyond his capabilities. This difference in the inherent powers of the liver in different persons is analogous to variations in their muscular and mental powers, and the less powerful should not be described as suffering from functional disease of their muscles or brain because they fail to accomplish the work which their better developed companions have no difficulty with. If, therefore, an individual consumes an amount of food that is excessive for his powers of digestion, fermentation and auto-intoxication will result. These poisons will impair the functional

[1] Brunton, L. *Clin. Journ.*, 1899-1900, xv, 177.

activity of the liver, and as a result the poisonous products of digestion will be allowed to pass into the general circulation and give rise to the various toxic manifestations already referred to. From what has gone before, it is evident that the well-known symptoms ascribed to a "torpid" or "inactive liver" are chiefly due to factors which secondarily interfere with the functional activity of the liver and not to a primary functional failure on the part of that organ. But because the ingenious conception of lithaemia and other views as to primary functional disease of the liver do not commend themselves in the light of later knowledge, it does not follow that hepatic insufficiency or inadequacy is a negligible factor. It is quite possible that primary functional disorder of the liver does occur, but in the present state of our knowledge its existence is almost impossible to recognise with accuracy.

The symptoms of secondary functional disturbance of the liver have already been referred to (p. 38).

Treatment.—The treatment of the symptoms of secondary hepatic inadequacy must therefore be directed to the causes and not to the liver itself. In the first place, the alimentary canal should be cleared out; this is most satisfactorily effected by the use of calomel, grs. iij., or of the old-fashioned blue pill, grs. v., followed by haustus sennae, or saline purgatives, such as sulphate of magnesium or sodium, phosphate of sodium, or some natural mineral water with purgative properties. The mercury drives the bile out of the gall-bladder, unloads the bile-ducts, and by sluicing the common duct tends to remove the causes of catarrh of its lower end. At the same time it acts as an intestinal antiseptic and inhibits excessive fermentation, and thus puts a stop to further auto-intoxication. The saline removes the mercury from the intestinal surface and prevents prolonged irritation, while at the same time it diminishes portal engorgement. The purgative action of these two remedies removes poisons from the alimentary canal. Intestinal fermentation and putrefaction can be most satisfactorily prevented by careful dieting and by the administration of minute doses of calomel ($\frac{1}{40}$ to $\frac{1}{20}$ grain) three times a day, which is preferable to salol, β-naphthol, and other popular drugs employed for this purpose. It is, of course, important that the bowels should be kept properly open. The ordinary purgatives already mentioned may be used for this purpose, or a pill containing euonymin and iridin. These two drugs are often employed and spoken of as if they had some special action other than that of purgatives, but without any satisfactory reason.

Plenty of water should be taken so as to wash out the poisonous products from the circulation and stimulate the functional activity of the kidneys. It is better not to take water in considerable quantities either with or directly after food, but to take it half an hour or so before a meal. Hot water may be sipped first thing in the morning and last thing at night. During the existence of symptoms a liquid diet, of which milk is the staple, should be adopted, and alcohol in any form

should be rigidly avoided. In the second place, the patient should be warned to avoid the forms of food likely to set up intestinal catarrh and fermentation. The articles of food that must be avoided as indigestible will, of course, vary in individual cases, but, generally speaking, the following should be forbidden: Concentrated and highly spiced soups; rich fish, such as salmon, mullet, eels, kippered fish; duck, hare, made dishes, entrées, pickles, rich sauces, melted butter, tea cakes, crumpets, sweets, cream, cheese, and much protein food. Alcohol should be avoided or taken in great moderation, well diluted, and with meals. Claret, hock, or whisky are the forms that may be taken if it is thought desirable, but beer, stout, cider, champagne, sherry, Madeira, Port, Burgundy, and liqueurs should be strictly prohibited.

Exercise is important. The form of exercise most suitable to individual requirements varies somewhat, and a stereotyped direction cannot be given to all cases. When the symptoms have existed for a considerable time and when muscular debility is present, active exercise is unsuitable, at any rate at first, and may lead to exhaustion and exaggeration of the symptoms. In such circumstances massage may give very good results and may be followed by carefully regulated exercise, such as Ling's Swedish gymnastics. In ordinary cases, however, massage and gymnastics are hardly necessary. In some persons walking briskly is sufficient, but in many, probably in the majority, a more active form of exertion is more effective, such as horse-riding, bicycling, lawn tennis, or golf. There is the additional advantage that these forms of exercise distract the attention from the individual's private business or worries. Open-air exercise is better than indoor, but when the weather is bad, fencing, racquets, boxing, or gymnastics may be of great use in improving the conditions of health.

It is important to get the skin to act, and for this purpose Turkish baths or hot baths with vigorous friction of the skin by rough towels are useful. When the skin has been acting vigorously after exercise the underclothing should be changed as soon as the individual comes in, so as to avoid chilling the surface of the body. The body should be well but not too warmly clad, and care must be taken to avoid chills to the legs, abdomen, and neck.

A visit to a spa, either in this country, such as Harrogate, Llandrindod Wells, or Strathpeffer, or abroad, Homburg, Ems, Neuenahr, Vichy, Carlsbad, Marienbad, may be followed by improvement or cure. The patient not only undergoes a carefully regulated course of treatment ("the cure"), but gets change, holiday, and rest from the cares of business and other worries.

As to the **prognosis**, the digestive disturbances which give rise to these symptoms are much the same as those leading to cirrhosis, and indeed the symptoms of functional disease of the liver may in some instances be the early manifestations of cirrhosis. As a rule, however, the prognosis is good provided the patient conforms to medical advice.

DISEASES OF THE HEPATIC ARTERY

Aneurysm; Embolism; Thrombosis, Arteriosclerosis, etc.

ANEURYSM

ANEURYSM of the hepatic artery is rare; Rolland[1] in 1907 referred to 41 instances.

Age and Sex.—In [illegible] 63 cases in which the ages were given, the average age was thirty-~~seven~~ eight years, the extremes being eighty-three and fourteen years. In 65 [illegible] cases, [illegible] 13 were females and [illegible] 53 males. The average age in the women was [illegible] 9 years higher (44 years) than in the men (35) ~~(Rolland)~~.

Causation. — Some of these aneurysms may be due to embolism, and from the comparatively unsupported condition of the visceral arteries in the abdominal cavity, simple non-infective embolism is more likely to be followed by aneurysm there than in other situations, except, perhaps, in the circle of Willis, where the arteries lie on a yielding water-bed, the subarachnoid space. Trauma has been noted as a cause of hepatic aneurysm; thus, Mester's[2] patient was kicked in the abdomen by a horse. In most cases of hepatic aneurysm there is no evidence of embolism or trauma, and yet these factors seem more probable than mere chronic endarteritis. Perhaps in some instances the cause of the embolism, such as a calcareous plate from the aorta, has naturally been overlooked, or has passed away before the patient's death. Grunert[3] suggests that many cases of hepatic aneurysm are due to some infective disease such as osteomyelitis, enteric, pneumonia. Out of 22 cases analysed by him some acute infection occurred shortly before the aneurysm appeared in 16. Often, however, no antecedent condition, except endarteritis, is recorded.

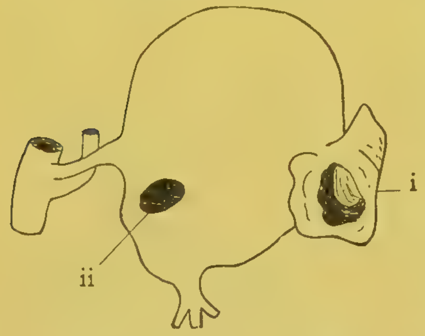

FIG. 10.—Aneurysm of hepatic artery, adherent to small intestine (i.), rupture (ii.) into the abdominal cavity. St. George's Hosp. Museum, Series vi, 86A. (Drawn by Dr. C. K. McKerrow.)

An aneurysm the size of a walnut was found by Pearson Irvine[4] inside an abscess in the left lobe of the liver; here the aneurysm was produced by inflammation and ulceration of the outer coats of the artery in the same way that aneurysms are formed in the vomicae of pulmonary tuberculosis.

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[1] Rolland, W. *Glasgow Med. Journ.*, 1908, lxix, 342.
[2] Mester. *Ztschr. f. klin. Med.*, 1895, xxviii, 93.
[3] Grunert. *Deutsche Ztschr. f. Chir.*, Leipz., 1904, lxxi, 158.
[4] Irvine, Pearson. *Trans. Path. Soc.*, Lond., 1878, xxix, 128.

ndenwald and Tannenbaum
~~[illegible]~~ in 1922 collected 65
~~[illegible]~~ cases

Mc Crae 1920. in 56

Hepatic aneurysm ~~appears~~ has been said to be unique in that
acute infection is more important etiologically than syphilis, but
Friedenwald & Tannenbaum, however, ascribed it in equal proportion to both,

ierdenwald and TANNENBAUM
~~[illegible]~~., Amer. Journ. Med. Sc., Phila., 1923, CLXV, ~~[illegible]~~ 11

Käding, K. Deutsche Ztschr. f. Chir., Leipz., 1919. CL, 92

In 18 out of ~~[illegible]~~ 65 cases there was an intrahepatic aneurysm ~~and in 4 of these the tumor ruptured into the peritoneal cavity (Rolleston)~~

In size the aneurysm varies from that of a millet seed to a child's head, but is usually about the size of a pigeon's egg.

Fever was noted in 16 out of 49 cases and may accompany the attacks of colic ^

Aneurysms of the hepatic artery or its branches may be due to ulceration in the gall-bladder or bile-ducts eroding the wall of the artery. At first an aneurysm would result, and later, by extension of the ulceration, the aneurysm would open into the biliary tract.

This ulceration may be due to gall-stones. Naunyn[1] regards Lebert's case of rupture of an hepatic aneurysm into the gall-bladder of a woman aged thirty, with fatal gastro-intestinal haemorrhage, as due to gall-stones, and quotes M. B. Schmidt's[2] case of ulceration of the bile-duct, in connexion with an impacted gall-stone, opening into an aneurysm of the hepatic artery.

In connexion with the production of hepatic aneurysm by ulceration starting in the gall-bladder or bile-ducts and possibly due to cholelithiasis, it is significant that in ~~[illegible]~~ 63 cases of hepatic aneurysm in which the sex is noted ~~[illegible]~~ 13 were in women and ~~[illegible]~~ 50 in men ~~(Rolland)~~. Aneurysm is so rare in women, while gall-stones are so common, that it is probable this proportion of female cases in aneurysm of the hepatic artery may to some extent be due to the mechanism of ulceration of the artery from without. Usually ~~(in 33 out of Rolland's 41 cases)~~ there is a single aneurysm on the trunk or extrahepatic branches of the hepatic artery, but in some instances more than one or even multiple aneurysms have been recorded. Aneurysm of the hepatic artery due to tuberculous infection and possibly the result of erosion by a tuberculous gland was described by Bickhardt and Schümann.[3]

In a boy aged seventeen there were two intrahepatic aneurysms (Borchers[4]), and in a boy aged eighteen Hale White[5] found an aneurysm on each of the main branches of the hepatic artery, one of which was embedded in the substance of the liver. Multiple aneurysms of small size may occur in great profusion in the liver in the rare disease periarteritis nodosa. From an analysis of 19 cases of the allied condition of polyarteritis acuta nodosa, Carnegie Dickson[6] finds that the liver is affected in nearly all the cases. ~~Intrahepatic aneurysms have, however, attracted very little attention, and are very rarely seen~~.

Symptoms.—Pain is nearly always present, and may be mistaken for that of biliary colic; in fact "pseudo-biliary" colic may be due to the pressure of the aneurysm on the bile-ducts. ~~The attacks may be accompanied by fever.~~ Jaundice ~~is frequently present~~ Occurred in more tha half the recorded cases and is usually due to pressure on the ducts; it may be slight or not appear until late in the course of the disease; when it comes on shortly before death, it may be due to leakage of the aneurysm into the ducts and the formation of a clot (Villandre[7]). ~~Rolland stated that it was present in 10 out of 41.~~

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[1] Naunyn. *On Cholelithiasis*, p. 141. Translation, New Sydenham Soc., 1896.
[2] Schmidt. *Deutsch. Arch. f. klin. Med.*, Leipz., 1893–4, lii, 536.
[3] Bickhardt und Schümann. *Ibid.*, 1907, xc, 288.
[4] Borchers. *Aneurysma d. Art. hepat.*, Kiel, 1878.
[5] Hale White. *Brit. Med. Journ.*, 1892, i, 223.
[6] Dickson, Carnegie. *Journ. Path. and Bacteriol.*, Cambridge, 1907, xii, 50.
[7] Villandre. *Arch. gén. de chir.*, Paris, 1909, iii, 111.

cases. Reichmann[1] says it occurred in 64 per cent of 39 collected cases. Aneurysm and new-growth in the portal fissure behave, in a miniature fashion, just as the corresponding lesions in the anterior mediastinum do with regard to the adjacent venous trunks. Aortic aneurysm rarely obstructs the superior vena cava, while mediastinal growth frequently does. In the same way aneurysm of the hepatic artery, though it may push the portal vein aside, does not obliterate it or give rise to ascites.

Hepatic aneurysm may perforate into the peritoneum (33 ~~17~~ out of ~~33~~ cases ~~(Rolland)~~), and give rise to fatal collapse. With about equal frequency ~~(13 out of 33 cases)~~ it ruptures into some part of the bile passages, and the blood enters the alimentary canal and may be vomited, or more often passed by the bowel alone; repeated leakage and haemorrhages may occur before a fatal one. The aneurysm may rupture into the hepatic or common bile-ducts, the gall-bladder, portal vein, or stomach. Acute pancreatitis has resulted from regurgitation of bile into Wirsung's duct, the ampulla of Vater being blocked by clot (Dean and Falconer).[2]

A tumour has hardly ever been felt, and pulsation has not been detected.

Hepatic aneurysm, as in a case recorded by Ledieu, may obstruct the circulation in its branches. Reichmann and Dean and Falconer record necrosis of the liver cells with the formation of cysts due to this cause. By experimental ligation of the hepatic artery in dogs Dujarier and Castaigne[3] found that the flow of bile is retarded and that infection of the bile-ducts is thus rendered more easy. This may explain cases in which multiple abscesses in the liver are associated with an aneurysm of the hepatic artery.

Ross and Osler[4] recorded a case in which the sac of an hepatic aneurysm became infective and multiple emboli passed into the liver, leading to numerous abscesses.

Prognosis is very unfavourable.

Diagnosis.—The diagnosis of hepatic aneurysm is very difficult. Most of the recorded cases have been regarded as duodenal ulcer or cholelithiasis. It would be very difficult to distinguish with certainty between an hepatic aneurysm and one of the abdominal aorta in the immediate neighbourhood; especially as the latter may press on the bile-duct and give rise to jaundice.

Treatment is usually merely palliative. Of 10 ~~6~~ cases treated surgically, 3 ~~5 died; in the one which~~ recovered ~~Kehr[5] successfully ligated~~ the hepatic artery and removed the aneurysm.

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[1] Reichmann. *Virchows Arch.*, 1908, cxciv, 71.

[2] Dean and Falconer. *Edin. Med. Journ.*, 1912, viii, 124.

[3] Dujarier et Castaigne. *Bull. Soc. anat.*, Paris, 1899, lxxiv, 329.

[4] Ross and Osler. *Canada Med. and Surg. Journ.*, 1877, vi, 1.

~~[5] Kehr, H. *München. med. Wchnschr.*, 1903, xl, 1861.~~

correct diagnosis was made in 2 out of 65 cases.

[illegible]
Mc Graw quotes a successful case operated upon
by [illegible] Dixon

From an experimental investigation and review of ligature of the hepatic artery SEGALL concludes that the production of infarction depends on the site of the ligature, and that the nearer the ligature is to the bifurcation of the arteria hepatica propria the more likely is necrotic infarction to occur. 1

SEGALL, H.N. *Surg., Gyn., and Obstet.*, Chicago, 1923, xxxvii,

EMBOLISM

Embolism of the main trunk of the hepatic artery is very rarely observed; possibly it is sometimes missed, because the artery is not systematically examined. Chiari[1] recorded embolism of the main trunk, and Lancereaux[2] and Ogle,[3] embolism at the bifurcation of the hepatic artery. In Lancereaux's case hepatic pain set in ten hours before death and was referred to the embolism. In Ogle's case there were anaemic infarcts in the liver, but no general necrosis of the liver. Necrosis of the liver has been recorded by Lancereaux and by Chiari in man, and experimentally embolism of the hepatic artery in the hands of Cohnheim and Litten,[4] and of Doyon and Dufourt,[5] has led to the same result. ^ ~~Experimental ligature of the hepatic artery produces necrosis in rabbits but not in dogs; Dujarier and Castaigne found that aseptic ligature in dogs was not followed by necrosis, but that when infection occurred necrosis resulted~~. Obstruction of the artery may favour portal thrombosis, and by retarding the flow of bile render infection of the bile-ducts easier. In Lancereaux's cases of obstruction of the hepatic artery the liver became greatly engorged from the absence of the driving power or *vis a tergo* normally supplied by the hepatic artery. ~~In rare cases~~ infarction of the liver has followed embolism. (*Vide* p. 105.)

In haemic infections embolism of the small branches of the hepatic artery gives rise to pyaemic abscesses. Embolism also occurs in generalised tuberculosis and sarcomatosis, especially in melanotic sarcoma.

THROMBOSIS

This is also a pathological curiosity. Lancereaux[6] described a case of a clot in the trunk of an atheromatous hepatic artery in a man aged sixty-five who died with arteriosclerosis and gangrene of the feet which may have been either embolic or thrombotic.

ARTERIOSCLEROSIS

The trunk of the hepatic artery is sometimes, in common with the rest of the arterial system, found to be atheromatous. When affected, the hepatic artery usually shews endarteritis deformans rather than endarteritis obliterans, or is dilated and varicose from loss of elasticity rather than narrowed. It thus disposes to the rare events, aneurysm and thrombosis of the hepatic artery. It is noticeable that arteriosclerosis does not lead to a fibrotic atrophy of the liver in any way resembling a granular arteriosclerotic kidney, though senile atrophy of the liver with some fibrous replacement might be thought to depend on a similar process. Inside the liver the branches of the hepatic artery shew endarteritis in the neighbourhood of syphilitic gummas. In haemochromatosis the hepatic artery shews endarteritis; occasionally in ordinary cirrhosis there is endarteritis obliterans. Bonome[1] describes haemorrhagic and necrotic infarcts due to endarteritis obliterans in cirrhosis. In general paralysis of the insane, Angiolella[2] has observed endarteritis of the hepatic artery.

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[1] Chiari. *Ztschr. f. Heilk.*, 1898, xix, 507.
[2] Lancereaux. *Traité des maladies du foie et du pancréas*, p. 541, 1899.
[3] Ogle, C. *Trans. Path. Soc.*, 1895, xlvi, 72.
[4] Cohnheim und Litten. *Virchows Arch.*, 1876, lxvii, 153.
[5] Doyon et Dufourt. *Arch. de physiol.*, 1898, 5. s., x, 522.
[6] Lancereaux. *Loc. cit.*, p. 543.

ENLARGEMENT OF THE HEPATIC ARTERY

Enlargement of the hepatic artery is seen in cases of new-growth—though not in all instances—in cases of portal thrombosis of some standing, and in ordinary cirrhosis.

In a case in which the liver weighed 14 pounds from carcinomatous infiltration, secondary to a primary growth in the oesophagus, the hepatic artery was dissected out and was small in size.

DISEASES OF THE HEPATIC VEINS

Occlusion (due to Thrombosis, Stricture); Chronic Periphlebitis and Endophlebitis; Suppurative Phlebitis; Embolism.

OCCLUSION

OCCLUSION of the hepatic veins may be due to thrombosis or stricture. The various ways in which occlusion is brought about will be considered first, and then the clinical features.

Thrombosis of the hepatic veins is somewhat rare; it is usually secondary to some other hepatic lesion, but may spread from the inferior vena cava. It may be set up in the following ways:—

(1) New-growth in the liver may extend into the hepatic veins and give rise to thrombosis.

(2) "Adenomas" in nodular hepatitis may rupture into the hepatic veins and thus lead to thrombosis.[3]

(3) In portal cirrhosis thrombosis of the hepatic veins is rare. It may, as has just been pointed out, occur in cases of cirrhosis with multiple adenoma or nodular cirrhosis. Further, thrombosis of the hepatic veins may be due to intercurrent acute or subacute infections

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[1] Bonome. *Arch. di biol.*, Firenze, 1899, liii, 319.

[2] Angiolella. *Manicomio mod.*, 1894, 1895.

[3] Compare Delépine. *Trans. Path. Soc.*, Lond., 1890, xli, 362.

(Parkes Weber, Jacobson and Goodpasture) ^

Parkes Weber. Proc. Roy. Soc. Med., 1915, VIII, (Med. Sect.) 6

Jacobson and Goodpasture. Arch. Int. Med., Chicago, 1918, XXII, 86.

falling on a cirrhotic liver and setting up inflammation which involves the thin walls of the hepatic veins. But in some cases of thrombosis of the hepatic veins with a slight degree of cirrhosis, the latter may be merely a secondary fibrous replacement.

(4) Thrombosis may be due to stricture of the hepatic veins. On the other hand, it has been argued that thrombosis may be followed by endophlebitis and stricture (Thompson and Turnbull, Fisher,[1] *vide* p. 51).

(5) Thrombosis may extend from the inferior vena cava.

Thrombosis of the inferior vena cava due to malignant disease of the kidney or adrenal may spread into the hepatic veins. In a case of thrombosis of the inferior vena cava due to perforation of an amoebic hepatic abscess, the hepatic veins contained thrombi (Winternitz[2]). I have seen a parietal thrombus in the inferior vena cava, where it grooves the liver, associated with thrombosis in the hepatic veins; there was more recent clot in the portal vein. In a case of obliteration of the inferior vena cava recorded by Reynaud the right hepatic vein was thrombosed. In a case of obliteration of the inferior vena cava reported by Dixon Mann and Hall[3] there were thrombi in the hepatic veins. In this case peritonitis was thought to have set up periphlebitis and endophlebitis and the subsequent changes in the inferior vena cava and hepatic veins.

Osler[4] ~~has~~ described considerable stenosis of the orifices of the hepatic veins in a case of obliteration of the inferior vena cava, a condition which would readily dispose to thrombosis.

(6) Thrombosis of the hepatic veins may also occur in association with widespread thrombosis.

Pitt[5] records thrombosis in the aorta, in the splenic, left renal, and right middle cerebral arteries, and in the right hepatic vein. Softened and therefore older thrombi were found in the portal vein.

It appears probable that thrombosis of the hepatic veins may be due to the same causes that set up portal thrombosis, or to an extension of that process.

(7) It may not be possible to assign a definite cause for thrombosis of the hepatic veins in all cases. Possibly toxins or micro-organisms stopped by the liver may be responsible for some cases. Fisher reports a case of hepatic vein thrombosis in a child with bronchopneumonia. Thrombosis of the hepatic veins occurs at an early stage in suppurative inflammation due to hepatic abscess, pylephlebitis, etc.

Stricture of the hepatic veins chiefly occurs at or close to their junction with the inferior vena cava. Pressure from tumours, hydatid cysts, or cicatricial adhesions may obstruct branches of the hepatic veins in the liver and lead to local venous engorgement (*vide* p. 86). Stenosis of the main trunks is rare. It may be due to the following causes:—

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[1] Fisher, T. *Bristol Med.-Chir. Journ.*, 1902, xx, 209.
[2] Winternitz. *Johns Hopkins Hosp. Bull.*, Balt., 1911, xxii, 396.
[3] Dixon Mann and W. Hall. *Edin. Med. Journ.*, 1904, N.S., xvi, 56.
[4] Osler. *Journ. Anat. and Phys.*, 1879, xiii, 291.
[5] Pitt, G. N. *Trans. Path. Soc.*, 1895, xlvi, 75.

(1) Cicatricial contraction starting outside the veins, either in the liver or in its neighbourhood. Under the name of phlebitis hepatica adhaesiva, Frerichs [1] described great narrowing of the hepatic veins due to inflammation spreading in from dense adhesions in the neighbourhood. His case was in an alcoholic married man aged forty-five; no mention of syphilis is made. He had ascites and jaundice; there were perihepatitis and probably some cirrhosis as well as a nutmeggy condition of the liver. Kelynack [2] met with a similar condition in a woman aged thirty-two who had probably had syphilis. There were adhesions around the hepatic veins, with marked stenosis and thrombosis; the adhesions were thought to have produced the stenosis.

(2) Gummatous disease or syphilitic fibrosis in the immediate neighbourhood of the main hepatic veins. A gumma may be so situated as to lead to obliteration and thrombosis in one or both of the hepatic veins. Fagge [3] refers to a case in which one of the hepatic veins was so much narrowed by the pressure of a gumma that it only admitted a probe. In rare instances a gumma has involved the inferior vena cava and obliterated the openings of the hepatic veins (Wilks,[4] West [5]).

(3) Extensive hepatic fibrosis and cirrhosis. In some cases the cicatricial formation is very extensive and is associated with general hepatic fibrosis. There is not in all cases a certain syphilitic history, but there is sufficient evidence to suggest that congenital syphilis is a probable factor in the general and extensive fibrosis.

In a man aged twenty-six years under Churton's [6] care ascites suddenly set in, and death occurred nine days later from exhaustion. Well-marked cirrhosis of the liver thought to be due to congenital syphilis with thrombosis and thickening of the walls of the hepatic veins was found at the necropsy; there was no portal thrombosis. Cicatricial contraction causing thrombosis of the hepatic veins was found in a boy aged thirteen who died in St. George's Hospital with well-marked cirrhosis of the liver. The patient had never taken alcohol, but there was reason to believe he was the subject of congenital syphilis, as his mother had three miscarriages after his birth and as there was marked fibrosis in the spleen, which weighed 16 ounces. The liver, 42 ounces, was considerably enlarged and most extensively fibrosed; a large mass of cicatricial tissue involving the hepatic veins suggested early gummatous change; no caseation, however, could be seen microscopically.[7]

The following case is probably a combination of syphilitic cirrhosis with endophlebitis obliterans: Gee [8] described complete obliteration of the orifices of the hepatic veins in a child aged seventeen months. Ascites rapidly appeared

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[1] Frerichs. *Diseases of the Liver*, ii, 432-437, New Sydenham Soc., 1861.

[2] Kelynack. *Med. Press and Circ.*, 1897, cxiv, 633.

[3] Fagge. *Principles and Practice of Medicine*, vol. ii, p. 295, 1886.

[4] Wilks, S. *Trans. Path. Soc.*, 1862, xiii, 123.

[5] West, S. *Ibid.*, 1891, xlii, 155.

[6] Churton. *Ibid.*, 1899, l, 145.

[7] Lazarus-Barlow, W. S. *Ibid.*, 1899, l, 147. St. George's Hosp. Museum, Ser. ix, 174 L.

[8] Gee. *St. Barth. Hosp. Rep.*, 1871, vii, 144.

at the age of fourteen months, and paracentesis was twice performed. The liver (16¾ ounces) was cirrhotic. There was considerable perihepatitis around the orifices of the hepatic veins. The hepatic veins ended abruptly, just short of entering the inferior vena cava, being cut off from it by a thin membrane. The lining membrane of the vena cava where the mouths of the hepatic veins should have been shewed dimples which had not at all the look of scars. Gee did not think this was due to a congenital failure of union of the venae revehentes hepaticae with the inferior vena cava, for then the ductus venosus should have remained open. He regarded the primary change as cirrhosis which led to stenosis and obliteration of the hepatic veins. Some of the hepatic veins contained firm clot. The liver was nutmeg. There was a free collateral circulation especially around the left branch of the portal vein. Similar cases have been described by Rosenblatt[1] and Penkert.[2]

(4) To primary obliterative endophlebitis of the hepatic veins. In a few cases there is marked or almost complete stenosis of the openings of the hepatic veins into the inferior vena cava without any evidence that the inflammatory process has spread to the veins from adjacent parts. Craven Moore[3] collected 12 and Hess[4] 23 cases of this primary obliterative inflammation of the hepatic veins. But they both, especially Hess, include cases in which the inflammatory process may have started from without such as Gee's, Rosenblatt's. It may be extremely difficult to decide whether the process started in or outside the veins. The openings into the inferior vena cava may be mere dimples. In some instances, as in two of Chiari's three cases,[5] syphilis was regarded as responsible, but this does not hold good in all cases. The entrance of the ductus venosus into the inferior vena cava in the immediate neighbourhood of the hepatic veins suggests that the process of obliteration of that fetal vessel may spread to the orifices of the hepatic veins and, by excess of the obliterating process, gradually lead to stenosis of the orifices of the hepatic veins, just as stricture of the ileum sometimes occurs at the point where Meckel's diverticulum is normally obliterated, or coarctation of the aorta where the ductus arteriosus joins the aorta. Moore, however, believes that congenital influences may so reduce the resistance of the mouths of the hepatic veins as to enable some haematogenous poison to induce endophlebitis. Kretz[6] suggests that the obliteration is secondary to small tears of the walls of the hepatic veins due to coughing, and that exuberant cicatrisation follows. Thompson and Turnbull[7] argue that the process is not a pure endophlebitis but a thrombophlebitis, and that thrombosis is likely to occur at the junction of the hepatic veins and inferior cava. In their two cases the

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[1] Rosenblatt. *Virchows Jahresb. der Med.*, Berlin, 1867, i, 267.

[2] Penkert. *Virchows Arch.*, 1902, clxix, 337.

[3] Craven Moore. *Med. Chron.*, 1902, xxxvi, 240.

[4] Hess. *Am. Journ. Med. Sc.*, Phila., 1905, cxxx, 986.

[5] Chiari. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1899, xxvi, 1.

[6] Kretz. "Pathologie der Leber," *Lubarsch-Ostertag Ergebnisse allg. Path.*, 1904 (1902), viii, Abth., ii, 498.

[7] Thompson and Turnbull. *Quart. Journ. Med.*, Oxford, 1911–12, v, 276.

diaphragmatic closure of the hepatic veins was ascribed to organisation of blood-clot.

Morbid Anatomy.—The liver is in a state of advanced chronic venous engorgement with dilatation of the trunks of the hepatic veins behind the stenosis. ‸ The Spigelian lobe may be greatly enlarged (Hess).

Clinical Features.—The symptoms are those of portal obstruction. Ascites is almost constant and pain in the hepatic region common. The veins over the abdomen may be enlarged. Vomiting is common, but haematemesis is rare, and jaundice most exceptional. The liver is usually enlarged and tender; and there may be cyanosis. The disease may either last for several months and imitate cirrhosis or portal thrombosis; or the symptoms may be very acute, beginning with rapid ascites, followed by delirium and coma, and terminating fatally in a few days. ‸ The disease may occur at any age, but the average age of Hess' 23 cases was 28·5 years. The sexes are equally affected. A correct diagnosis has never been made. The prognosis is extremely bad.

CHRONIC PERIPHLEBITIS AND ENDOPHLEBITIS OF THE HEPATIC VEINS

In adherent pericardium inflammation around the inferior vena cava may spread to the hepatic veins and set up periphlebitis and possibly slight fibrosis in the neighbourhood. In the cases that I have examined, however, there has been no extension of inflammation from the walls of the veins into the surrounding liver substance. In long-standing backward pressure the intima of the hepatic veins may, like that of the inferior vena cava, become somewhat thickened and opaque—a slight degree of chronic endophlebitis due to increased intravascular pressure, and the veins become dilated. Endophlebitis may spread from the inferior vena cava in cases of obliteration of that vessel into the hepatic veins, and may, as in Dixon Mann and Hall's[1] case, give rise to thrombosis of the hepatic veins.

SUPPURATIVE PHLEBITIS OF THE HEPATIC VEINS

Suppurative phlebitis is more likely to attack the hepatic veins than the branches of the portal vein in hepatic abscess, since the latter are surrounded by Glisson's capsule, while the hepatic veins and their branches are not protected in this way. When the hepatic veins are thus affected, general pyaemia is more likely to occur than in pylephlebitis, since the emboli can pass more readily into the circulation.

In a case of large hepatic abscess in a man aged thirty-four the abscess set up thrombosis in the right hepatic vein which produced embolism of the right pulmonary artery and suppurating areas in both lungs. In a case of suppuration around a calculous gall-bladder recorded by West[2] the abscess opened into the hepatic veins and produced multiple infarcts in the lungs.

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[1] Dixon Mann and W. Hall. *Edin. Med. Journ.*, 1904, N.S., xvi, 56.

[2] West, S. *Trans. Path. Soc.*, Lond., 1886, xxxvii, 281.

^| The centre of the lobule may shew necrosis.

Sudden hepatic enlargement coinciding with the onset of acidosis in a case with signs of obstruction of the Inferior vena Cava may show that the hepatic Veins have become acutely obstructed (Jacobson and Goodpasteur).

In a case of proctitis, due to a bacillus of the influenza group, Ophüls[1] found abscesses in the submucous coat of the rectum, areas of necrosis in the liver, and suppurative thrombosis of the hepatic veins which had produced secondary foci in the right lung. The portal vein was quite healthy. In an areolar abscess of the liver due to *Bacillus mucosus capsulatus* the suppuration started in connexion with the hepatic veins (Hewitt[2]).

Embolism of the Hepatic Veins

Embolism of the hepatic veins can occur only when the embolus travels against the blood-stream and enters the hepatic veins from the inferior vena cava—in other words, retrograde embolism. Retrograde embolism of veins is very rare; when it occurs, it is more frequently seen in the hepatic veins, since they are not protected by valves, and are so close to the heart that fragments of growth or thrombus may drop into their orifices either from the inferior vena cava or from the heart and superior vena cava. Welch[3] quotes examples of fragments of new-growth in the hepatic veins in cases in which the primary growths were in the abdomen and thyroid.

It seems probable that in cases of cranial suppuration with secondary abscesses in the liver, without any abscesses in the lungs, the micro-organisms may drop down the jugular vein, superior vena cava, right auricle, and inferior vena cava into the orifices of the hepatic veins, and so infect the liver. The production of retrograde embolism probably depends on the temporary stagnation or reversal of the direction of the blood-flow. Thus, if a thrombus was passing up the inferior cava and a violent expiratory effort or cough occurred at the moment when it was opposite the openings of the hepatic veins, the embolus might be carried into the liver.

THROMBOSIS OF THE PORTAL VEIN

Synonyms: Pylethrombosis, Pylephlebitis Adhaesiva.

In this condition there is thrombosis of the portal vein which does not go on to suppuration.

Causation.—It will be convenient to deal first with thrombosis depending upon inflammatory and other morbid conditions of the portal vein, and then to consider the influence of hepatic cirrhosis, intra-abdominal malignant disease, tumours, adhesions, and trauma in the production of portal thrombosis.

Inflammatory Conditions causing Portal Thrombosis.—Thrombosis of the portal vein, or, as it may in this particular connexion be more appropriately called, adhesive pylephlebitis, occurs as a preliminary stage in suppurative pylephlebitis. Whether in a given case the process goes on to suppuration or not probably depends on the nature and virulence of the organisms. In 48 cases analysed by Langdon Brown[1] there was an infective origin in 7, or 14·6 per cent. By extension from adjacent parts inflammation may spread to the walls of the portal vein and set up thrombosis, which does not necessarily go on to suppuration. This may occur in cholangitis, in pancreatitis, in peripancreatitis, in hepatic and subphrenic abscesses, a suppurating mesenteric gland, and in other conditions.

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[1] Ophüls. *Am. Journ. Med. Sc.*, 1901, cxxii, 797.
[2] Hewitt. *Johns Hopkins Hosp. Bull.*, Balt., 1909, xx, 77.
[3] Welch, W. H. Allbutt's *System of Medicine*, 1899, vi. 232.

Cholelithiasis, by setting up cholangitis and pericholangitis, may lead to inflammation of the walls of the portal vein and subsequently to thrombosis. In Lissauer's[2] 68 cases of portal thrombosis there were 9 with gall-stones, Kanis[3] described portal thrombosis secondary to an ulcer in the common bile-duct without gall-stones. Extension of inflammation from an hepatic abscess is a rare cause of thrombophlebitis of the portal vein.

A man aged twenty-seven, who had contracted dysentery in the Boer War of 1899–1902, was brought in a moribund state into St. George's Hospital. At the necropsy there was acute peritonitis with several pints of turbid fluid in the abdominal cavity, due to rupture of an abscess in the left lobe of the liver into the peritoneal cavity. There was a firm dry clot in the portal vein, which might have given rise to the ascites. The walls of the portal vein were greatly thickened and the lymphatic glands in the portal fissure much enlarged. It seemed probable that the infection had spread, at any rate partly, by the lymphatic vessels from the abscess to the trunk of the portal vein.

Inflammation, abscess, or infarction of the spleen may set up thrombosis of the splenic vein, which may spread into the portal vein. *Simple ulcer of the stomach* or duodenum must be a very rare cause of portal thrombosis. Wickham Legg[4] and KASPAR mention~~s~~ ~~two~~ cases; ~~but I have no other references~~. Thrombosis of the gastric veins may cause ulceration of the gastric mucous membrane. I have seen this in a case of suppurative pylephlebitis due to appendicitis. Indirectly a gastric ulcer may cause portal thrombosis by giving rise to adhesions compressing the portal vein, or to a localised abscess in the neighbourhood. I have seen portal thrombosis in a case of subphrenic abscess due to perforation of a gastric ulcer.

Probably in many of the cases that appear to be primary thrombosis there is in reality microbic infection of a low grade of virulence present which is responsible for the production of a thrombus. The term "adhesive pylephlebitis," formerly used to designate thrombosis of the portal vein, is an expression of the view, originated by Hunter and Cruveilhier, that thrombosis is always the result of phlebitis. Bacteriological examination[5] proves that many thrombi which would

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[1] Brown, Langdon. *Brit. Med. Journ.*, 1905, ii, 1393.
[2] Lissauer. *Virchows Arch.*, 1908, cxcii, 278.
[3] Kanis. *Wien. klin. Rundschau*, 1911, xxv, 757.
[4] Legg, Wickham. *St. Barth. Hosp. Rep.*, 1874, x, 236.
[5] Vaquez, *Thèse de Paris*, 1890; Bryant, J. H., *Guy's Hosp. Rep.*, 1902, lvi, 99.

A) And among 37 Cases of Cholangitis [illegible]
found 4 or 10.8 per cent of portal thrombosis

KASPAR, F. Deutsche Ztschr. f. Chir., 1920, CLVI, 1

formerly have been regarded as marantic contain micro-organisms, and though the presence of micro-organisms does not necessarily prove that they caused the thrombosis, it justifies the return to the view that most thromboses are secondary to infection of the wall of the vein.

Disease of the Portal Vein.—Chronic phlebosclerosis, which is probably often due to increased blood-pressure in the portal vein, plays an important part in producing portal thrombosis. Though generally associated with cirrhosis, chronic portal phlebitis may be independent of any hepatic disease. It is quite possible that phlebosclerosis may follow thrombosis; but it seems probable that it usually precedes and disposes to portal thrombosis. Borrmann,[1] who insisted on this sequence of events, found phlebosclerosis in 7 out of 20 cases of portal thrombosis.

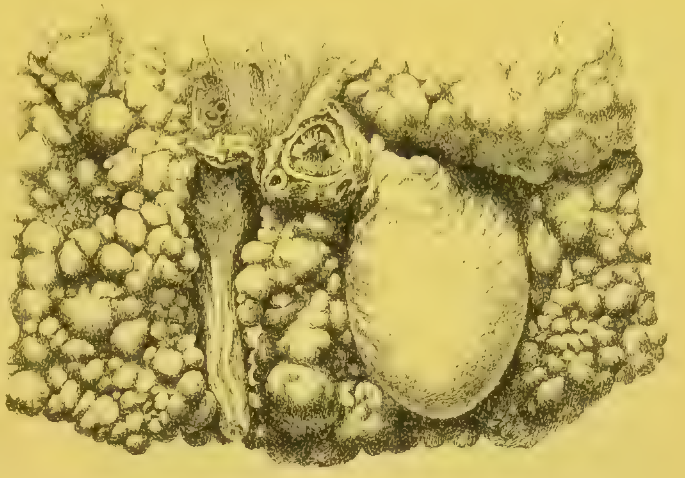

FIG. 11.—The under surface of a cirrhotic liver, shewing thrombosis of the portal vein. (Drawn by Dr. E. A. Wilson.)

When the change is of old standing calcification of the wall of the vein may occur, and calcareous plates or spicules may project into the lumen of the portal vein and induce thrombosis.

In a patient on whom I made a necropsy at St. George's Hospital, the dependence of thrombosis on calcification of the portal vein seemed clear. A man aged sixty-six years, addicted to some alcoholic excess, had been in good health until two months before his death, when ascites appeared. He was thin, but there had been no melaena or haematemesis. Paracentesis was required, but the fluid reaccumulated, diarrhoea set in, and he became delirious and died comatose. At the necropsy there were pigmented scars in the colon as if from old dysentery, but no ulceration. There was localised fibrinous peritonitis around the site of the trocar punctures. The portal vein was much thickened and shewed calcification, and there was a softening thrombus in the right branch of the portal vein. The splenic vein was occluded at its entry into the portal vein and contained a crumbling thrombus in contact with an area of calcification in its wall. The thrombosis did not correspond to the more extensive changes in the walls of the portal vein, and it therefore appeared probable that the thrombosis was secondary to the changes in the portal vein, as presumably the thrombosis had only existed since the ascites developed, viz. two months. The liver was atrophied, weighing 32 ounces, but not cirrhotic. The spleen weighed 18 ounces and on section appeared fibrotic, but did not present any infarcts.

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[1] Borrmann, *Deutsch. Arch. f. klin. Med.*, Leipz., 1897, lix, 283.

Syphilitic inflammation of the portal vein probably gives rise to thrombosis in some cases. This may occur in both the hereditary and the acquired forms of the disease. In 68 cases of portal thrombosis 7, or 10 per cent, were associated with syphilitic disease of the liver (Lissauer).

Cirrhosis of the Liver.—Although cirrhosis is probably the most frequent cause of portal thrombosis it does not appear to be present in half the total cases of that condition.

In 60 cases collected from various sources, including most of Langdon Brown's, there were 22, or 36·7 per cent, due to cirrhosis. These 22 cases do not include those in which cirrhosis and intra-abdominal malignant disease were both present, or the few examples in which the observers believed cirrhosis to be secondary to portal thrombosis. In Lissauer's 68 cases of thrombosis cirrhosis was present in 6 only.

Portal thrombosis is far from common in cirrhosis of the liver.

In a period of thirty-three years Langdon Brown[1] found that in 334 necropsies performed on cases of hepatic cirrhosis at St. Bartholomew's Hospital 10, or 3·3 per cent, were complicated by thrombosis of the portal vein. In 711 cases of cirrhosis Lissauer found pylethrombosis in 6, or ·9 per cent.

In cirrhosis of the liver there are a number of factors favouring portal thrombosis, such as obstruction to the passage of blood through the liver and stagnation of blood in the vein, while the increased venous pressure tends to set up endophlebitis and even secondary calcification of the walls of the vein. Further, the catarrh of the intestinal tract, so common in portal cirrhosis, favours microbic invasion of the walls of the tributaries of the portal vein. In nodular cirrhosis, or cirrhosis with multiple adenoma, thrombosis of the portal vein is relatively common; thus, in 15 cases collected by Ll. Powell[2] there was portal thrombosis in 9. The contents of the adenomas may soften down from necrosis and discharge into the portal vein, thus setting up thrombosis. On the other hand, thrombosis of the portal vein may so impair the nutrition of the adenomas as to set up fatty degeneration and necrosis.

Intra-abdominal Malignant Disease.—After cirrhosis the commonest condition associated with portal thrombosis is malignant disease either in the liver itself or elsewhere in the abdominal cavity. In 60 cases of portal thrombosis 10 were associated with some form of intra-abdominal malignant disease.

Malignant Disease of the Liver.—Carcinoma of the liver may eat its way into the branches of the portal vein and induce thrombosis; this may occur either in primary or secondary malignant disease of the organ, but it is particularly prone to occur when primary carcinoma supervenes

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[1] Brown, Langdon. *St. Barth. Hosp. Rep.*, 1901, xxxvii, 62.

[2] Powell, Ll. *Unpublished Thesis for M.B. Cantab.*, 1895.

Among 275 [illegible] necropsies on cases of cirrhosis at the Johns Hopkins Hospital there were 7 examples of portal thrombosis or 2·6 per cent. (WEBSTER).

Webster, L.T. Bull. Johns Hopkins Hosp., Baltimore, 1921, xxxii, 16.

In 100 cases of malignant disease ∧
of the liver (9 primary, 91 secondary)
the portal vein was thrombosed
in 4, 2 in primary and 2 in secondary
malignant disease (Anderson).

in a cirrhotic liver. In these cases the contents of the portal vein may be composed of growth as well as of blood-clot.

In other forms of intra-abdominal disease, such as carcinoma of the stomach, pancreas, colon, the growth may involve the tributaries of the portal vein, spread along them, and set up thrombosis.

Carcinoma of the Stomach.—Malignant disease of the stomach occasionally gives rise to portal thrombosis. In Lissauer's 68 cases of portal thrombosis malignant disease of the stomach was present in 7, or 10 per cent. S. and S. Fenwick[1] found it in 3 per cent of their cases of gastric carcinoma. Gastric cancer may set up portal thrombosis in several ways; the growth may enter the gastric veins and extend directly into the portal vein and cause secondary thrombosis; pass up the lesser omentum by continuity and compress and involve the portal vein; or by means of secondary growths in the portal fissure, ~~or~~ in the substance of the liver, or near the head of the pancreas may compress the portal vein.

In the following case there were at least two factors favouring thrombosis of the portal vein. A woman aged sixty-five years died in St. George's Hospital with a small cirrhotic liver weighing 28 ounces and a spheroidal-celled carcinoma near the pylorus. The trunk of the portal vein was thickened and surrounded by adhesions. The right branch of the portal vein was thrombosed and there was an adherent clot at the entrance of the splenic vein. In a case of portal thrombosis secondary to gastric carcinoma Longcope[2] found infarcts in the liver. In a case of carcinoma of the lower end of the oesophagus in a man, examined after death at St. George's Hospital, there was a subphrenic abscess in connexion with breaking down carcinomatous glands, and as a result of the abscess thrombosis of the portal vein.

Malignant Disease of the Pancreas.—In 41 cases of portal thrombosis Langdon Brown found that the condition was associated with malignant disease of the pancreas in three instances. Sometimes when a malignant growth compresses the portal vein, and also infiltrates its walls, a finger-like process of the growth may extend along the lumen of the vein and then set up a secondary thrombosis. This very rarely happens in malignant disease of the pancreas; ~~it is chiefly seen in malignant disease in the liver~~.

Malignant disease of the glands in the portal fissure secondary to malignant disease of the stomach, pancreas, liver, or gall-bladder might compress the portal vein. Malignant disease of the liver may compress as well as invade branches of the portal vein, and set up thrombosis which travels distally into the main trunk. Portal thrombosis has been recorded in primary carcinoma of the bile-ducts (Bourgeret and Cossy[3]). In Lissauer's 68 cases of portal thrombosis there was primary carcinoma of the gall-bladder in 6, or 9 per cent.

Pressure on the portal vein, by tumours, adhesions, and altered conditions of adjacent viscera, may be, but is not necessarily, accompanied by thrombosis of the portal vein. By pressure the nutrition of the walls of the vein is impaired, microbic infection is thus favoured, and stagnation of the blood is induced—factors which all dispose to thrombosis. Pressure may be exerted on the portal vein in a number of ways, chiefly by malignant disease involving the adjacent lymphatic glands or the head of the pancreas.

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[1] Fenwick. *Cancer and other Tumours of the Stomach*, 1902, p. 72.

[2] Longcope. *Univ. Penn. Med. Bull.*, Phila., 1901, xiv, 223.

[3] Bourgeret et Cossy. *Bull. Soc. anat.*, Paris, 1873, xlviii, 347.

Walker[1] records occlusion and thrombosis of the portal vein by fibrous adhesions due to calculous pericholecystitis.

Chronic Pancreatitis.—The cicatricial contraction of chronic pancreatitis may greatly constrict the superior mesenteric vein or the commencement of the portal vein and lead to thrombosis. Barnard[2] met with a case of this kind. Probably many cases of chronic pancreatitis were formerly spoken of as "scirrhus" of the pancreas.

Chronic peritonitis in rare instances may be the only other morbid lesion forthcoming, and so appear to be the cause, either by extension of inflammation to the walls of the portal vein, or, in very rare instances, by constricting or compressing the vein.

In St. Bartholomew's Hospital Museum there is a specimen [No. 2205 A] of thrombosis associated with chronic peritonitis; the liver was not cirrhotic. Frerichs quotes similar cases.

The traction exerted by adhesions due to gastric or duodenal ulcers (Frerichs[3]) may compress the portal vein and in rare instances induce thrombosis. Similarly adhesions involving the mesenteric veins may start thrombosis which may extend upwards into the trunk of the portal vein (F. Taylor,[4] R. Johnson[5]).

The mechanical pressure exerted by a calculus, in the gall-bladder or more frequently in the ducts, on the portal vein may give rise to simple thrombosis. This is more likely to occur when the calculus is in the common duct, where it is in close contact with the portal vein.

Cases are recorded by Leudet, Geigel, Naunyn,[6] Westenhöffer,[7] Körte.[8] Naunyn also describes a case in which a calculus in the cystic duct compressed and gave rise to portal thrombosis, and refers to a case of Klesser's in which a calculus in the same position compressed one branch of the portal vein. Donkin[9] met with portal thrombosis due to pressure exerted by several calculi in the gall-bladder on the vein.

Gummatous inflammation in the portal fissure is a rare cause. It is possible that *aneurysm* of the abdominal aorta or of the hepatic artery may press on the portal vein and induce thrombosis.

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[1] Walker. *Ann. Surgery*, London, 1907, xlv, 27.

[2] Barnard, H. L. *Clin. Journ.*, 1905, xxvi, 133.

[3] Frerichs. *Diseases of the Liver*, i, 272, Transl. New Sydenham Soc., 1860.

[4] Taylor, F. *Trans. Path. Soc.*, 1881, xxxii, 21.

[5] Johnson, R. *Trans. Clin. Soc.*, 1898, xxxi, 212.

[6] Naunyn. *Cholelithiasis*, Transl. New Sydenham Soc., p. 133, 1896.

[7] Westenhöffer. *Semaine méd.*, 1903, xxiii, 32.

[8] Körte. *Ibid.*

[9] Donkin. *Med. Press and Circ.*, 1868, ii, 396.

Trauma may be followed by portal thrombosis; cases have been described by Heller,[1] Ponfick, Saxer, Schmorl,[2] Pitt.[3] As an example of post-operative thrombosis Delatour's[4] case of thrombosis of the splenic vein after splenectomy, extending into the portal vein, may be mentioned. Trauma involved in volvulus or in kinking of the mesentery may be the starting-point of thrombosis in the portal area (Langdon Brown).

Thrombosis of the portal vein may be secondary to *thrombosis of the hepatic veins.* The portal thrombosis is probably terminal. Cases have been described by Chiari,[5] Thompson and Turnbull,[6] and myself.[7]

Puerperal eclampsia has been thought by Schmorl[8] to induce portal thrombosis. This may be the result of degenerative changes in the liver giving rise to discharge of necrotic hepatic cells and tissue fibrinogens into the portal vein.

In a number of cases there is no local cause or associated morbid change, such as cirrhosis or inflammation, to account for portal thrombosis. Of this group of *primary portal thrombosis* Lewis and Rosenow[9] have collected 21 cases. In some of these cases it is associated with thrombosis elsewhere in the body, and therefore almost certainly depends on a widespread change in the blood of either a chemical or infective nature. It is analogous to the thrombosis sometimes seen in marasmus. The altered blood condition must favour infection, and the intestinal tract would appear to be so eminently favourable to the passage of micro-organisms into the tributaries of the portal vein that it is surprising that portal thrombosis is so comparatively infrequent. I have on two occasions seen portal thrombosis associated with thrombosis in the iliac veins. Langdon Brown found that 6 of 33 cases were associated with thrombosis elsewhere. Portal thrombosis in a girl aged 18 years was associated with and possibly favoured by an abnormality of the course of the portal vein (Hecht[10]).

It is curious that nothing is known as to the occurrence of portal thrombosis in connexion with diseases such as gout, influenza, pneumonia, and especially typhoid fever, which lead to thrombosis elsewhere in the body.

Sex.—More cases occur in males than in females. In 62 cases, 38 were males and 24 females; the preponderance of males depends on the greater frequency of hepatic cirrhosis in that sex. Cirrhosis of the liver was present in 18 out of the 38 males and in only 4 of the 24 female cases. Intra-abdominal malignant disease accounted for 8, or exactly one-third of the cases of portal thrombosis in women.

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[1] Heller. *Verhandl. d. deutsch. path. Gesellsch.*, Jena, 1904, p. 182.
[2] Schmorl. *Ibid.*, Karlsbad, 1902, p. 150.
[3] Pitt, G. N. *Trans. Path. Soc.*, London, 1895, xlvi, 74.
[4] Delatour. *Ann. Surg.*, 1895, xxi, 24.
[5] Chiari. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1899, xxvi, 1.
[6] Thompson and Turnbull. *Quart. Journ. Med.*, Oxford, 1911–12, v, 281.
[7] Rolleston. *Trans. Path. Soc.*, 1899, l, 148.
[8] Schmorl. Quoted by Welch, Allbutt's *System*, 1899, vi, 219.
[9] Lewis and Rosenow. *Arch. Int. Med.*, Chicago, 1909, iii, 233.
[10] Hecht. *Wien. klin. Wchnschr.*, 1908, xxi, 944.

Age.—In 61 cases the average age was forty-four years. The extremes were eighty years in a man and five years in a girl.[1] In the two sexes the average ages were 44·8 years among the males and 41 years among the females. This again corresponds with the slightly earlier age at which cirrhosis is fatal in women. There were ten patients under thirty years of age, equally divided among the two sexes; four were under twenty years of age—three females and one male. The average age of portal thrombosis is thus more than twelve years above the average age (31·6 years) in suppurative pylephlebitis. The explanation of this is that the two conditions—hepatic cirrhosis and intra-abdominal malignant disease—which most frequently cause portal thrombosis are commoner after forty years of age, whereas appendicitis, which is the most prolific source of suppurative pylephlebitis, is essentially a disease of young persons.

The distribution of the thrombosis in the portal vein varies considerably. It may extend throughout the portal vein and its branches. Usually it is more limited; it may occupy the main trunk of the portal vein and one or both of its branches in the liver, or it may obstruct the trunk and one or more of its tributaries, whilst in other instances thrombosis may be confined to one of its intrahepatic branches or one of its tributaries. The clot obstructing a tributary, such as the splenic vein, may just project into, but not obstruct, the blood-flow through the main trunk of the portal vein; in many instances the clot extends from the tributaries upwards into the portal vein. In other instances the clotting may begin in the portal vein and subsequently spread into the peripheral tributaries. Thrombosis may be confined entirely to the tributaries of the portal vein—the splenic, superior and inferior mesenteric veins—or to its intrahepatic branches.

Morbid Anatomy.—The vein is distended with clot, and thus differs from the more or less collapsed condition of the portal vein when containing ordinary post-mortem blood-clot. Distal to the obstructing thrombus the vein may be considerably dilated or even sacculated; this may also occur in the splenic vein. The vein wall is usually thickened, sometimes considerably so, from chronic phlebosclerosis, a condition analogous to arteriosclerosis, which Borrmann considers may be responsible for thrombosis. The wall of the vein sometimes contains calcareous plates, a further stage of chronic phlebosclerosis. In other instances the vein wall is swollen from recent inflammation. If the patient lives for a long time after the onset of thrombosis the portal vein may be reduced to a mere cord of fibrous tissue. In such cases a collateral circulation develops (p. 209), and a plexus of veins may take the place of the portal vein. Dilated veins in the lesser omentum may convey blood to the liver—a hepatopetal collateral circulation (Pick[2]). ∧ It is possible that in some cases thickening and calcification of the walls of the portal vein are results of thrombosis with partial canalisation and organisation of the blood-clot.

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[1] Taylor, F. *Trans. Path. Soc.*, 1881, xxxii, 61.

[2] Pick, L. *Virchows Arch.*, 1909, cxcvii, 490.

The coronary vein of the stomach may
be greatly dilated (GRUBER)

GRUBER. Mitt. a.d. Grenzgeb. d. Med. u. Chir., JENA, 1912, XXV, 735

The character of the clot varies according to its age: it may be quite recent and not very firmly adherent to the vein wall, or it may be granular, decolorised, or even laminated. In some instances the lumen of the vein is not obliterated, the clot being only parietal. As has already been mentioned, the thrombosis may be parietal in one part of the portal vein and completely obstruct the lumen in another, or the central part of the clot may be canalised. When the central part of the thrombus softens down, the condition approaches, if it does not merge into, suppurative pylephlebitis. The thrombus may obstruct, without spreading into, the orifice of one of the tributaries of the portal vein; this is not uncommon at the junction of the splenic vein with the trunk of the portal vein.

The *liver* may present conditions that have caused or favoured the occurrence of thrombosis in the portal vein, such as portal cirrhosis, nodular cirrhosis, primary or secondary malignant disease, or abscess. The liver may shew changes secondary to portal thrombosis. Thus, the hepatic artery may be enlarged in order to compensate for obstruction to the advent of blood to the liver by means of the portal vein. Thrombosis of the portal vein may be associated with a fatty or necrotic condition of the "hobnails" of a cirrhotic liver, and it may reasonably be believed that portal thrombosis, by cutting off the blood-supply, has brought about this change. It is, however, possible that the converse occurs, and that a softened hobnail may, by discharging into a branch of the portal vein, start thrombosis.

Infarcts, though rare in the liver, are, when present, associated in a fair percentage of the cases with portal thrombosis. They are, however, very far from being a necessary sequence of that condition. Some other factor, such as thrombosis in the intralobular branches of the portal vein, obstruction of the hepatic artery, or toxaemia, is necessary before infarction occurs in portal thrombosis (*vide* p. 106). The softening and necrosis of cirrhotic hobnails in portal thrombosis might be compared with the production of an anaemic infarct in an otherwise healthy liver under similar conditions. In portal thrombosis the infarcts are usually red or haemorrhagic, and in only a few instances anaemic.

Frerichs[1] described local atrophy and depressions, sometimes leading to a lobulated condition of the liver, as a result of obstruction of individual branches of the portal vein. It is possible that in an early stage of these areas of atrophy there was an infarct.

When portal thrombosis has existed for some considerable time the liver may shew very little change, or may merely be fatty.

In a remarkable case recorded by Langdon Brown[2] symptoms of portal obstruction had existed for twenty years, and indeed this diagnosis had been made early in the patient's illness by the late Sir William Jenner. The portal vein was represented by a fibrous cord with a minute lumen. The liver was described as natural though pale. Similar cases have been recorded by Cruveilhier,[1] Versé,[2] Winternitz.[3] In other instances the liver is small, atrophied, and shews a little replacement fibrosis. In a case regarded as primary thrombosis of the portal vein by Dickinson[4] in a woman aged twenty-one, the liver, though fatty, presented no signs of cirrhosis and weighed only 28 ounces. A similar case was recorded by Peacock.[5] In a case which I examined after death, the liver weighed 46 ounces and appeared atrophied to the naked eye. Microscopically there was no cirrhosis, though the fibrous tissue around the portal areas was very prominent. I have found exactly the same appearances in two other cases.

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[1] Frerichs. *Diseases of the Liver*, ii, 396. Transl. New Sydenham Soc., 1861.

[2] Brown, Langdon. *St. Barth. Hosp. Rep.*, 1901, xxxvii, 64.

It has been thought by West[6] and Goodhart[7] that thrombosis of the portal vein may give rise to portal cirrhosis in a previously healthy liver. There is ample evidence that this need not occur; on the other hand, some atrophy of the liver cells with fibrous replacement is not uncommon, and this relative fibrous increase might have been regarded as cirrhosis of slight degree.

Fibrosis of the liver was produced in dogs by Solowieff[8] as a result of gradual thrombosis of the portal vein. This might possibly have been due to some extension of inflammation into the liver along the portal vein, ~~or have been a replacement fibrosis associated with atrophy of the liver cells from diminished blood supply~~. Bermant's[9] experimental researches support Solowieff's views as to the production of cirrhosis by portal obstruction; Bainbridge and Leathes[10] found that ligature of a branch of the portal vein in animals produced atrophy of the liver cells and some replacement fibrosis; Cohnheim and Litten[11] came to a diametrically opposite conclusion.

To sum up the late effects of portal thrombosis on the liver, there may be no alteration except slight fatty change; there may be atrophy of the liver with some fibrous replacement, but there is very little evidence that genuine cirrhosis is produced in this way.

Spleen.—Enlargement of the spleen is almost constant when the trunk of the portal vein is completely occluded by a thrombus. Langdon Brown estimates that it is enlarged in 70 per cent of the cases. Enlargement may be reduced by copious ascites or gastro-intestinal haemorrhages, or prevented by firm perisplenic adhesions or chronic capsulitis. In a case under my care of thrombosis ~~of the~~ portal ~~vein~~ in cirrhosis associated with rapid and excessive ascites, the spleen weighed 4 ounces only.

On the other hand, there may be no splenic enlargement with a

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[1] Cruveilhier. *Atlas d'Anat. pathol.*, livraison xvi, Pl. 6.
[2] Versé. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1910, xlviii, 520.
[3] Winternitz. *Johns Hopkins Hosp. Bull.*, Balt., 1911, xxii, 396.
[4] Dickinson. *Trans. Path. Soc.*, Lond., 1863, xiv, 63.
[5] Peacock. *Ibid.*, 1873, xxiv, 122.
[6] West, S. *Ibid.*, 1888, xxxix, 106.
[7] Goodhart. *Ibid.*, 1889, xl, 134.
[8] Solowieff. *Virchows Arch.*, 1875, lxii, 195.
[9] Bermant (1897). Quoted by Welch, Allbutt's *System*, 1899, vi, 221.
[10] Bainbridge and Leathes. *Bio-chemical Journ.*, Liverpool, 1906, ii, 25.
[11] Cohnheim und Litten. *Virchows Arch.*, 1876, lxvii, 153.

Rous and LARIMORE found that ligature of branches of the portal vein in rabbits and dogs caused atrophy without fibrosis in the corresponding area of the liver, but the ~~hyp~~ atrophy was conditional on hypertrophy, and if proliferation of the liver cells was prevented by ligature of the bile duct atrophy did not ~~occur~~ progress.

Rous and LARIMORE. Journ. Exper. Med., Baltimore, 1920, xxxi, 609.

Worthen
intermediate [illegible]

McGee
has a figure

parietal thrombosis of the portal vein or where the mesenteric veins only are obstructed. Very great enlargement of the spleen follows complete thrombosis of the splenic vein; it may weigh 20 or 30 ounces and may shew infarcts,[1] either anaemic or haemorrhagic. In connexion with the association of infarcts and thrombosis of the splenic vein it should be remembered that an infarct due to arterial embolism may induce thrombosis.

Intestines.—When there is extensive thrombosis of the mesenteric veins there may be haemorrhagic infarction going on to gangrene in part of the small intestine, usually in the middle of the jejunum which is more often affected than the rest of the intestine because it is entirely dependent for its blood-supply on the superior mesenteric artery and does not draw any blood from other sources, such as the parietal veins around the duodenum and caecum. From gangrene of the intestine peritonitis may result.

~~I have seen~~ *secondary ulceration of the stomach* may be due in portal thrombosis. When the portal vein has been obstructed for some time, an extensive collateral circulation develops, and the small veins in the intestines, oesophagus, lesser omentum, and stomach may be considerably dilated. A varicose vein in the stomach may become eroded and give rise to fatal haemorrhage. In a case of portal thrombosis due to phlebosclerosis, in which the liver was not cirrhotic, fatal haematemesis occurred from an ulcerated oesophageal varix (Curtis [2]).

Clinical Picture.—*Onset.*—In the presence of cirrhosis of the liver or intra-abdominal disease there may be nothing to mark the occurrence of portal thrombosis, especially when ascites is already present. In latent cirrhosis or other cases in which the patient has been in good or in fair health, the onset may be sudden and marked by the rapid development of ascites or by profuse haematemesis. In 34 cases analysed by Langdon Brown the onset was gradual in 14 and acute in 20. Probably the onset is acute when the vein is suddenly occluded and gradual when it occurs slowly.

Symptoms and Signs.—The clinical manifestations of portal thrombosis present a certain amount of variation which may roughly be correlated (1) with the situation and extent of the thrombosis in the portal area, and (2) with the rate at which the lumen of the vein becomes obliterated. Thus, if the portal vein alone is involved, the most prominent symptoms are due to obstruction to the passage of blood through the liver, viz. ascites and haematemesis. If the thrombus occludes the proximal end of the splenic vein where it joins the portal vein, enlargement of the spleen and haematemesis may be expected. When thrombosis of the mesenteric veins occurs acutely it gives rise to haemorrhagic infarction of the mesentery and intestines, and is associated with intestinal obstruction, diarrhoea, melaena, and collapse.

Arranged in the order of their frequency and importance, the chief clinical features of portal thrombosis are: ascites, enlargement of the spleen, gastro-intestinal haemorrhages, abdominal pain, symptoms of intestinal obstruction, diarrhoea, and manifestations of toxaemia resembling those in the last stages of cirrhosis and not unlike uraemia. It will be seen that thrombosis of the portal vein may present the aspect of cirrhosis of the liver, of a gastro-intestinal disorder, or may combine the features of these two groups.

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[1] Rolleston. *Trans. Path. Soc.*, 1892, xliii, 49.

[2] Curtis. *Proc. Roy. Soc. Med.*, Lond., 1909, ii (Path. Sect.), 159.

Ascites is the most frequent clinical manifestation. In 61 collected cases it was present in 40, or 65·6 per cent. It may be absent in acute cases which rapidly prove fatal from gastro-intestinal haemorrhage, or when the mesenteric veins are chiefly involved and the symptoms suggest intestinal obstruction. Copious gastro-intestinal haemorrhage or diarrhoea may prevent the development of ascites. In the rare cases in which life has been prolonged for years there may be no ascites, presumably from compensation brought about by a free collateral anastomosis. Stress was laid by Frerichs on the rapid development of ascites and on its reaccumulating directly after paracentesis as an important diagnostic sign of portal thrombosis, but this is not of much value, as the same phenomena may occur in cirrhosis and occasionally in ascites due to other causes.

Enlargement of the Spleen.—The spleen is very frequently found enlarged after death, but is not palpable in all these cases during life. Since most statistics refer to the size of the spleen as determined after death, a much lower estimate than 70 per cent, the percentage arrived at by Langdon Brown by adding his 41 cases to Frerichs' and to 25 collected by Crofton-Atkins, must be made of the existence of a palpable spleen *intra vitam.* The enlargement may be masked by ascitic or tympanitic distension, and may diminish after copious gastro-intestinal haemorrhage. Very considerable enlargement should suggest the possibility of haematemesis or melaena due to a thrombus obstructing either the splenic vein or its junction with the portal vein. In chronic cases the splenic enlargement may closely imitate chronic splenic anaemia, especially when the splenic vein is thrombosed(Vide p. 86)

Gastro-intestinal symptoms are somewhat variable and are related to thrombosis involving the trunks or orifices of the gastric and mesenteric veins, rather than to thrombosis of the trunk of the portal vein. Gastro-intestinal haemorrhage was noted in 27 out of 61 collected cases, or in 44·2 per cent. Haematemesis and intestinal haemorrhage may be due to venous oozing or to definite ulceration from necrosis of the mucosa induced by thrombosis of the gastric or mesenteric veins. When the mesenteric veins are thrombosed, haemorrhagic infarction may occur and give rise to paralysis of the bowel with haemorrhagic effusion into its lumen. Gastro-intestinal haemorrhage may be the initial symptom; this was so in 7 out of 20 cases of acute onset collected by Langdon Brown. Haematemesis and melaena may be frequently repeated and rapidly prove fatal. On the other hand, in cases that survive for a number of years haemorrhages may recur from time to time during

A venous hum over compensatory varices has been reported (Hatzieganu and Sharteu). ^

The blood In chronic cases shows anaemia with a low colour index, and leucopenia, as in so called Banti's disease. Most exceptionally polycythaemia rubra ~~has been present~~ (van der Weyde and van Ijzeren) and eosinophilia (Hatzieganu and Sharteu) have been reported.

Van der Weyde et van Ijzeron. Semaine méd., Par., 1903, XXII
282

Hatzieganu et Sharteu. Bull. et mém. Soc. méd. des hôp. de Paris, 1924, 3e sér., xlviii, 947 the course of the disease in spite of a very considerable collateral circulation.

Diarrhoea.—Frerichs found that diarrhoea was present in 25 out of 28 cases, but in Langdon Brown's 41 cases it was noted in 11 only. The motions are watery and may be mixed with blood. There may be a somewhat sudden onset of acute intestinal symptoms, due to haemorrhagic infarction and paralysis of part of the small intestine.

Dilatation of the Superficial Abdominal Veins.—Enlargement of the subcutaneous veins around the umbilicus, resembling that in ordinary cirrhosis, is noted in less than one-third of the cases of portal thrombosis. It has been thought that its rapid appearance points to portal thrombosis, but no weight can be laid on this, or indeed on the presence or absence of dilated veins around the umbilicus.

Jaundice may be due to the cause responsible for the portal thrombosis; but it does not depend on portal thrombosis, and usually does not occur in uncomplicated cases.

In 41 cases it was present in 13 (Langdon Brown). It may be due to malignant disease, *e.g.* carcinoma of the head of the pancreas, compressing or obstructing both the portal vein and the bile-duct. In only one of Langdon Brown's cases was it thought that the thrombosed vein pressed on the common bile-duct.

The *urine* is diminished in amount. This may be explained as due to the lowered blood-pressure, since experimental ligature of the portal vein is followed by a great fall in arterial pressure. Other causes are loss of blood, diarrhoea, and diminished absorption of fluid. The urine is high coloured and lithatic. Alimentary glycosuria has been thought to be constantly present, and its absence to be good evidence that a suspected case is not one of portal thrombosis. Sugar, if absorbed from the intestinal tract, must pass by collateral channels straight into the general circulation, since it cannot reach the liver by the portal vein. The absorption from the intestines is greatly obstructed, and it is very doubtful if this test is of much value (see p. 234).

It was certainly absent one day before death in a patient with simple portal thrombosis, probably secondary to thrombosis of the hepatic veins; I have never observed it.

In a case of cirrhosis with thrombosis of the portal vein, Boinet[1] observed paralysis of the legs; he also produced paralysis of the hind limbs in animals by aseptic ligature of the portal vein.

In some cases the symptoms are those of toxaemia resembling uraemia, and probably due to hepatic insufficiency. The symptoms are therefore the same as in the terminal stage of portal cirrhosis.

I have seen widespread cutaneous and visceral haemorrhages in portal thrombosis.

Experimentally complete ligature of the portal vein at its entrance to the liver causes death (Bolton[1]); toxic symptoms, such as paraplegia, low temperature, and drowsiness, develop; there is also a marked fall of arterial blood-pressure which may be associated with the great engorgement of the portal system. Castaigne and Bender[2] refer death in experimental ligature of the portal vein to this factor and not to toxaemia. Incomplete obstruction of the lumen may be followed by survival, and if so there is ascites (Bolton).

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[1] Boinet. Quoted by Gorget, *Rev. de méd.*, 1897, xvii, 539.

Diagnosis is very difficult, and a correct opinion is therefore seldom reached during life. The sudden onset of ascites or of haematemesis and their recurrence, accompanied by considerable splenic enlargement, might suggest it. But since these manifestations are much the same as those of *cirrhosis*, with which portal thrombosis is so often associated, it is difficult to differentiate between portal thrombosis and cirrhosis. Cirrhosis is so common, whereas portal thrombosis is so comparatively rare, that in any given case the probabilities are in favour of cirrhosis, even though the onset of symptoms is sudden and severe. The sudden onset of ascites in a case of cirrhosis under my care suggested pylethrombosis, but the cause was *tuberculous peritonitis.*

In some cases of *gastric ulcer* profuse gastro-intestinal haemorrhage with collapse may suggest portal thrombosis. Difficulty is likely to occur only when the gastric ulcer is acute and occurs in an adult male or in a woman who has never had any signs of gastric ulcer and is considerably past the age at which it is commonly seen.

Very profuse haematemesis and melaena in a soldier, aged thirty-five years, coming on suddenly at Pretoria and accompanied by a low temperature, led me to an erroneous diagnosis of portal thrombosis; at the necropsy there was acute "diphtheritic" dysentery with an ulcer of similar nature in the stomach.[3]

In *splenic anaemia* the spleen is very considerably enlarged, there is anaemia of the chlorotic type, with a diminished number of leucocytes, and recurrent gastro-intestinal haemorrhages may occur. The disease is essentially chronic, whereas thrombosis of the portal vein is usually rapid. But in some instances of portal thrombosis, especially when the splenic vein is occluded, the spleen is greatly enlarged, and there may be periodic gastro-intestinal haemorrhages for many years, with fairly good health in the intervals.

Thus, Langdon Brown[4] quoted the case of a woman who had had haematemesis at intervals of ten months for twenty years, and in whom the portal and splenic veins were found occluded. Cases have also been recorded by Dévé,[5] and ~~some have~~ it has been thought that this is the real basis of many cases of chronic splenic anaemia.

Prognosis.—The diagnosis being so beset with difficulties the practical application of prognosis is very limited, but there is no doubt that the

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[1] Bolton, C. *Proc. Roy. Soc.*, London, Ser. B, 1907, lxxix, 267.

[2] Castaigne et Bender. *Arch. de méd. expér.*, Par., 1899, ii, 551.

[3] *The Imperial Yeomanry Hospitals in South Africa*, 1902, iii, 193 (Med[illegible]·and Surg[illegible]· Rep[illegible]·).

[4] Brown, Langdon. *St. Barth. Hosp. Rep.*, 1901, xxxvii, 155.

[5] Dévé. *Semaine méd.*, Par., 1908, xxviii, 212.

Dock and Warthin, Edens, Oettinger and Fiessinger.

Dock and Warthin. *Am. Journ. Med. Sc.*, Phila., 1904, cxxvij, 24

Edens. *Mitt. a. d. Grenzgeb. d. Med. u. Chir.*, Jena, 1907, xviij, 59.

Oettinger et Fiessinger. *Rev. de méd.*, Par., 1907, xxvij, 1109.

Chauffard et Troisier. *Presse méd.*, Par., 1913, xxxiv, 653

prognosis is infinitely better than in pylephlebitis. It is probable that if the process of portal thrombosis is gradual, so that a compensatory circulation can to some extent be developed before the obstruction becomes absolute, the patient has a much better chance of surviving.

If recovery from the acute symptoms occurs, the patient may live many years, even though the main trunk of the portal vein is obstructed. Thus, Langdon Brown, Osler,[1] Lyons,[2] Rogers,[3] and others have recorded cases in which life was prolonged for many years. In these cases the development of a collateral circulation to some extent compensates for the portal obstruction. There is, however, usually only a partial return to health, as from time to time haematemesis recurs and relieves the engorgement, whilst a very profuse gastro-intestinal haemorrhage or a succession of haemorrhages may prove fatal. ~~It is also possible that~~ thrombosis may occur in the vessels forming the compensatory circulation([illegible]

Treatment.—Haematemesis and ascites should be treated on the ordinary lines as in cirrhosis. When portal thrombosis is suspected, the coagulation time and the calcium content of the blood should be determined by A. E. Wright's[4] method, and if the coagulability of the blood and the amount of calcium salts are both found to be increased, citric acid in 30-grain doses may be given three times a day in order to prevent the process of thrombosis extending. The rationale of giving citric acid is to "decalcify" the blood and so reduce its coagulability. It is important that the blood should be tested before giving citric acid, since in some cases of cirrhosis, which is the morbid lesion most frequently associated with portal thrombosis, the alkalinity and coagulating power of the blood are diminished, and no good, and possibly harm, would result from citric acid. In syphilitic subjects iodides and mercury should be given on the chance that there may be a gumma pressing on the portal vein or syphilitic changes in the walls of the vein.

The operation of promoting vascular adhesions around the liver would theoretically be an ideal form of treatment for portal thrombosis, but there would probably be practical difficulties in performing it. Haemorrhage would be likely to occur from the engorged vessels, and there is the further danger that an extension of the thrombotic process might be induced by the necessary manipulation. This operation was performed in one case, but without benefit (Langdon Brown). In recurrent haematemesis thought to be due to portal and splenic thrombosis splenectomy has been suggested (Curtis[5]).

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[1] Osler, W. *Journ. Anat. and Physiol.*, 1882, xvi, 208.

[2] Lyons. *Dubl. Journ. Med. Sc.*, 1877, lxiv, 457.

[3] Rogers, B. M. H. *Bristol Med.-Chir. Journ.*, 1899, xvii, 107.

[4] Wright, A. E., and Knapp, H. H. *Lancet*, 1902, ii ; *Med.-Chir. Trans.*, 1903, lxxxvi, 1.

~~[5] Curtis. *Proc. Roy. Soc. Med.*, Lond., 1909, ii (Path. Sect.), 159.~~

SUPPURATIVE PYLEPHLEBITIS

Synonyms: Portal Pyaemia, Porto-pyaemic Liver Abscess (Davidson).

Suppurative inflammation may involve the portal vein together with some of its tributaries and the terminal branches in the liver; thus, in appendicitis there may be continuous suppurative pylephlebitis involving the superior mesenteric vein and the trunk and the intrahepatic branches of the portal vein. Usually it is less extensive and may be limited to the extrahepatic or to the intrahepatic branches. When the branches inside the liver are alone affected, the resulting condition is practically the same as multiple abscesses due to infective embolism of the branches of the portal vein; the only difference is one of degree, viz. more continuous purulent inflammation of the intrahepatic branches of the portal vein in intrahepatic pylephlebitis, while in multiple abscess the branches of the portal vein may be healthy for most of their extent. Since the clinical features of multiple hepatic abscesses due to intestinal infection are much the same as those of pylephlebitis, the description of the latter will cover that of multiple hepatic abscesses. Suppurative pylephlebitis is not common; it is less often seen than multiple liver abscesses due to infective emboli conveyed by the portal vein.

The infrequency of pylephlebitis is shewn by the following statistics. In twenty years 11 cases only were met with at Guy's Hospital (Bryant [1]); and in thirty-three years only 12 were found at St. Bartholomew's Hospital out of 9494 necropsies, or in 0·12 per cent (Langdon Brown [2]).

Etiology.—Suppurative infection of the portal vein is generally secondary to gastro-intestinal lesions, such as ulceration and suppuration.

Thus, in 64 cases of pylephlebitis tabulated by Langdon Brown, 45, or 70·3 per cent, were associated with gastro-intestinal lesions; of the remaining 19, no cause at all was forthcoming in 7; 4 were due to gall-stones; 2 were associated with empyema; and 6 with other forms of intra-abdominal suppuration.

As might naturally be expected from analogy, infection of the portal vein is most readily produced by a collection of pus confined under considerable tension. Thus, a localised abscess in connexion with appendicitis is a relatively frequent cause of suppurative pylephlebitis, while diffuse inflammation of the peritoneum, though it may be a result, is not by itself a causal factor. In some cases pylephlebitis may be secondary to suppuration in the substance of the liver, for example, in single or "tropical" abscess. Suppuration in the gall-bladder and bile-ducts may also cause infective pylephlebitis. A septic wound of the main trunk of the portal vein is hardly likely to cause pylephlebitis, as rapid

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[1] Bryant, J. H. *Guy's Hosp. Rep.*, 1900, liv, 77.

[2] Brown, Langdon. *St. Barth. Hosp. Rep.*, 1901, xxxvii, 95.

death from haemorrhage would probably follow. But an exploring syringe might infect some of the intrahepatic branches of the portal vein and so set up pylephlebitis. In a few carefully examined cases no inlet for the infection is found in the area of the portal vein.

In a case of suppurative pylephlebitis in which the primary disease was an empyema rupturing into the lung, Bryant could find no lesion of the alimentary canal, but he considered as possible explanations either that pyogenetic cocci in swallowed pus were absorbed from the stomach and thus reached the portal vein, or that exploratory punctures had passed through the empyema into the liver and thus directly infected it.

Possibly in some of the cases in which no definite source of inlet is found in the intestinal tract the cause may have been pyorrhoea alveolaris and the associated swallowing of pus and micro-organisms. The pyogenetic micro-organisms could pass through microscopic lesions in the stomach into the portal vein.

Appendicitis is the most frequent cause of suppurative pylephlebitis and multiple liver abscesses. This association has been emphasised by the term "appendicular liver" (Dieulafoy[1]). In 64 cases of suppurative pylephlebitis collected by Langdon Brown, the appendix was the source of infection in 27, or 42·2 per cent. But appendicitis is luckily only followed by suppurative pylephlebitis in about 0·5 per cent of the cases.

In 257 cases of perforative appendicitis Fitz[2] found suppurative pylephlebitis in 11 cases, and in 1189 cases of appendicitis operated upon in the Mount Sinai Hospital, New York, there were only 9 cases, or 0·75 per cent (Gerster[3]), whilst in 2714 clinical cases of appendicitis collected from various London hospitals[4] there were 10 cases of suppurative pylephlebitis, or ·4 per cent. In 86 fatal cases of appendicitis collected by Kelly and Hurdon[5] there were 10 cases of pylephlebitis and liver abscess; this high percentage may be due to such cases being published on account of this rare complication.

Infection of the portal vein is more likely to occur when an abscess has formed around the appendix and the pus is under pressure. Pylephlebitis may supervene very rapidly, so that it is well established by the time the appendicular abscess is opened.

In 1908 a man aged 22 was taken ill on June 25, an appendicular abscess was opened on July 4, but the temperature (*vide* Fig. 12) remained raised and he died on July 25 with pylephlebitis.

Sometimes the morbid process in the appendix has begun to subside when the body of a patient with pylephlebitis is examined. Pylephlebitis may occur after the appendix has been removed, infection of the mesenteric veins having already taken place.

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[1] Dieulafoy. *Clinique médicale de l'Hôtel-Dieu*, 1898, ii, 167; *Manuel de pathologie interne*, 1901, ii, 785.

[2] Fitz. *Trans. Assoc. Am. Phys.*, 1886, i, 110.

[3] Gerster. *Med. Rec.*, N.Y., 1903, lxiii, 1005.

[4] Statistics of Hawkins, Lett, Lockwood, Jones, and Clogg and Fairbank. *Med.-Chir. Trans.*, Lond., 1905, lxxxviii, 517.

[5] Kelly and Hurdon. *The Vermiform Appendix*, 1905, p. 225.

In a boy under the care of Mr. Pendlebury,[1] in St. George's Hospital, the appendix was removed on May 16 and the temperature remained normal until June 13, 1904, when it rose and a rigor occurred. The temperature continued raised until death, on July 8. There was extensive suppurative pylephlebitis.

Impaction of a pin in the vermiform appendix is rare, but when it does occur there is a great likelihood of intrahepatic suppuration; in 8 out of Mitchell's[2] 33 cases of impacted pins in the appendix there was some form of intrahepatic suppuration, in most instances multiple or pylephlebitic abscesses.

Gastric ulcer is very rarely the starting-point of suppurative pylephlebitis. Cases have been recorded by Carrington,[3] Bristowe,[4] Hart,[5] West,[6] Hawkins and Nitch[7] (3 cases), and others.

In several of the cases pylephlebitis was associated with, and probably the result of, localised suppuration set up by the gastric ulcer. Hart's case was complicated by a left pyopneumothorax. In Carrington's case a gastric ulcer had eroded the substance of the liver and set up local suppuration.

Gastric Carcinoma.—Suppurative pylephlebitis very seldom follows cancer of the stomach; it is even rarer than in simple ulcer. It may in rare instances be due to infection from a sloughing carcinoma. Legg[8] reported a case in carcinoma of the pylorus.

Disease of the Oesophagus hardly ever gives rise to pylephlebitis. Edwards[9] records suppurative pylephlebitis secondary to carcinoma 3 inches from the cardiac orifice.

Duodenal ulcer is a most exceptional cause of suppurative pylephlebitis; Warfvinge[10] and Bryant[11] have reported cases.

Intestinal Ulceration.—The two common forms of ulceration of the small intestine, typhoid and tuberculous, very seldom set up pylephlebitis. Possibly this is accounted for by the open condition of the ulcers, which allow free drainage and discharge into the bowel. When the inflammatory process is more circumscribed in the walls of the bowel, the radicles of the portal vein are in greater danger of infection. It is even rarer then than solitary hepatic abscess after typhoid fever.

Keen[12] refers to 5 cases only of pylephlebitis, Osler[13] has seen one case only, and no case has occurred either at St. Bartholomew's or Guy's Hospitals (Langdon Brown[14]) after typhoid fever.

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[1] Pendlebury. *Lancet*, 1904, ii, 1712.
[2] Mitchell. *Johns Hopkins Hosp. Bull.*, 1899, x, 35.
[3] Carrington. *Guy's Hosp. Rep.*, 1882, xli, 375.
[4] Bristowe, J. S. *Trans. Path. Soc.*, 1858, ix, 279.
[5] Hart, S. *Med. and Surg. Rep. Presbyterian Hosp.*, 1900, p. 150.
[6] West, S. *Trans. Path. Soc.*, 1890, xli, 146.
[7] Hawkins and Nitch, *Med.-Chir. Trans.*, 1907, xc, 339.
[8] Legg, W. *St. Barth. Hosp. Rep.*, 1874, x, 239; *Trans. Path. Soc.*, 1874, xxv, 123.
[9] Edwards. *Brit. Med. Journ.*, 1910, i, 199.
[10] Warfvinge. *Schmidt's Jahrb.*, 1882, cxcv, 130.
[11] Bryant. *Guy's Hosp. Rep.*, 1900, liv, 77.
[12] Keen, W. W. *Surgical Complications and Sequels of Typhoid Fever*, 1898, p. 247.
[13] Osler, W. "Studies in Typhoid Fever," *Johns Hopkins Hosp. Rep.*, No. 3, p. 381.
[14] Brown, Langdon. *Brit. Med. Journ.*, 1905, ii, 1393.

Dysentery very seldom gives rise to suppurative pylephlebitis. Bryant records one case. Infective emboli, however, may cause multiple hepatic abscess both in amoebic and in bacillary dysentery.

Penetration of the mesenteric veins by infecting foreign bodies is extremely rare; though a closely allied condition, pins perforating the vermiform appendix, is a less infrequent antecedent of suppurative pylephlebitis.

Lambron[1] published a case in which a fish-bone passed from the stomach into the superior mesenteric vein and set up pylephlebitis. Winge[2] recorded another instance, and Carlson[3] a case in which a fish-bone was supposed to have been carried from the vermiform appendix into the left lobe of the liver where it set up pylephlebitis.

S. Phillips[4] has described suppuration in the spleen and pylephlebitis due to bristles in the inferior mesenteric vein.

The following *pelvic conditions* occasionally give rise to suppurative pylephlebitis: operations on the rectum for piles, fissure, and stricture; carcinomatous and other forms of rectal ulceration; suppuration in connexion with the female genital organs, such as suppurating ovarian cysts, pyosalpinx (De Silva,[5] Bryant).

Suppuration of the umbilical vein in the newly born may extend into the liver and give rise to pylephlebitis (*vide* p. 571). Cantlie[6] states that this condition is extremely common in Hong-Kong.

Suppurative and gangrenous pancreatitis may be associated with suppurative pylephlebitis. Étienne[7] has insisted on the spread of inflammation from the pancreas to the portal vein and the production of suppurative pylephlebitis.

A man aged forty was in St. George's Hospital under the care of Mr. Turner, in 1899, with whom I often saw the patient. On admission there was a fluctuating abscess in the epigastrium which was at once opened, and found to come from the region of the pancreas. The patient improved for a time, but fever and rigors occurred; empyema on the left side and slight jaundice followed. At the necropsy the pancreas was gangrenous, there was pylephlebitis, and pus was found in the intrahepatic branches of the portal vein.

It may, however, be very difficult to decide whether the suppuration in the pancreas is the cause or the result of pylephlebitis. When there is some other available cause for pylephlebitis, such as appendicitis, suppuration in the pancreas must be regarded as secondary.

Suppurating Mesenteric Glands, Suppuration between the Layers of the Mesentery, etc.—Frerichs[8] describes cases, but the glandular affection may, at least in some instances, have been secondary to the suppurative pylephlebitis. In other cases the suppuration in the portal vein and in the mesenteric glands may be both concomitant results of some intestinal or appendicular ulcer that has been overlooked or has healed. J. W. Ogle[1] reported pylephlebitis secondary to suppuration in a collection of hydatid cysts connected with the omenta.

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[1] Lambron. *Arch. gén. de méd.*, Par., 1842, xiv, 129.
[2] Winge. *Schmidt's Jahrb.*, 1882, cxcv, 131.
[3] Carlson. *Hygiea*, 1907, 2. f., vii, 822.
[4] Phillips, S. *Trans. Clin. Soc.*, Lond., 1895, xxviii, 222.
[5] De Silva. *Ceylon Med. Journ.*, July 1890.
[6] Cantlie, J. *Encyclopaedia Medica*, 1901, vii, 41.
[7] Étienne. *Arch. de méd. expér. et d'anat. path.*, 1898, x, 177.
[8] Frerichs. *Diseases of the Liver*, New Sydenham Soc., 1861, ii, 421.

Abscess of the Spleen.—Cases have been recorded by Frerichs,[2] Law,[3] and Langdon Brown. It is probable that in some instances the splenic abscess is in reality secondary to pylephlebitis and not the primary cause. It seems more probable that perisplenic or subdiaphragmatic abscess on the left side may be a primary source of infection for pylephlebitis, as the pus is then under some pressure. It is possible that infective endocarditis by embolism of the branches of the splenic artery may lead to sloughing infarcts of the organ, and so to suppurative thrombosis of the splenic vein and pylephlebitis.

Suppuration in the Liver.—As a result of infection spreading from a solitary abscess or from a suppurating hydatid cyst secondary pylephlebitis may be set up. A suppurating hydatid is, however, much more often the cause of suppurative inflammation of the bile-ducts than of the portal vein, but may give rise to both. Suppurative cholangitis is frequently complicated by an extension of the inflammatory process to the intrahepatic branches of the portal vein, and it is highly probable that the infection may spread by the lymphatics. In most cases in which suppurative pylephlebitis complicates suppurative cholangitis it is evidently secondary and not nearly so widespread. But this tendency to pylephlebitis in suppurative cholangitis makes the latter disease even more formidable.

Gall-stones.—The usual mechanism by which cholelithiasis sets up multiple liver abscesses is by suppurative cholangitis; the multiple abscesses are then in connexion with the bile-ducts. Gall-stones in the ducts may, however, induce pylephlebitis, either by direct spread of inflammation from the ducts to the walls of the veins or through the lymphatic or blood-vessels. In rare cases a fistulous passage between a suppurating bile-duct and the portal vein, or one of its branches, may be the method by which gall-stones in the ducts may set up pylephlebitis; in exceptional instances the gall-stone may pass into the portal vein. It is often stated that three calculi were found in the portal vein of Ignatius Loyola, but this historically interesting case has been disputed by Thudichum[4] and Galliard,[5] the former asserting that they were phleboliths.

The lymphatics may become inflamed in cases of cholangitis, and in this way suppuration may extend along the portal spaces. The suppuration

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[1] Ogle, J. W. *St. George's Hosp. Rep.*, 1867, ii, 347.
[2] Frerichs. *Diseases of the Liver*, 1861, ii, 418, New Sydenham Soc.
[3] Law. *Dublin Quart. Journ.*, 1851, xi, 237.
[4] Thudichum. *A Treatise on Gall-stones*, p. 11, 1863.
[5] Galliard. *Méd. mod.*, Paris, 1895, vi, 113.

may then extend to the portal vein, and the pylephlebitis may supervene on cholangitis. This sequence of events probably occurred in a man aged forty-four whose case was described by Strangeways Pigg and myself.[1] In this case there was no evidence of gall-stones; but if it occurs in non-calculous cholangitis, it may presumably occur also in the calculous form. The infection may pass from the ducts by the small veins of the bile-ducts, which open into the branches of the portal vein. Bright[2] recorded a case of calculous cholangitis in which pylephlebitis was probably produced in this way. An abscess due to cholecystitis may erode the portal vein, discharge into it, and set up pylephlebitis and multiple suppurating foci in the liver (Bristowe[3]).

In some instances it is very difficult to make out how cholelithiasis gives rise to suppurative pylephlebitis. A calculus may give rise to cholangitis and infection may spread to the portal vein and so set up pylephlebitis, but the calculus in the meanwhile may be passed and the cholangitis may disappear.

The following case illustrates the difficulties that may arise: a man aged fifty-one was admitted to St. George's Hospital with a history of recent gall-stone colic; he was apparently going on fairly well until he was suddenly seized with abdominal pain and collapse. I saw him when *in extremis* and thought that probably perforation of an abdominal viscus had taken place. At the necropsy there was no perforation of the viscera or peritonitis, but there was suppurative pylephlebitis without any definite cause. The larger bile-ducts were carefully examined and no ulceration or calculi were found in them; the gall-bladder contained two calculi.

Sex.—The condition is commoner in males than in females, probably in accordance with the greater frequency of appendicitis in the male sex. In 76 cases, 52 were males and 24 females.

Age.—The disease usually occurs earlier in life than portal thrombosis; this may be correlated with the fact that it is most frequently secondary to appendicitis. When it occurs later in life it may be due to gall-stones or to malignant or other forms of ulceration of the colon.

In 60 cases the average age was 30 years; being the same in the 40 males and the 20 females. Langdon Brown found that half the cases occur between the ages of twenty and twenty-nine.

Morbid Anatomy.—The portal vein or its branches contain pus and broken-down blood-clot; sometimes there is merely pus, in other instances there is much blood-clot and but little pus, the infection and softening of the thrombus being only in an early stage. The vein walls are swollen, acutely inflamed, and from the resulting softening the vessels dilate, there are endo-, meso-, and periphlebitis; the walls may thus melt away in the suppurative process. As a result of the inflammation of the wall of the vein, blood-clot forms and becomes adherent to the intima. The clot softens down and a mixture of pus, clot, and blood results. This sanious pus may be of a reddish tint, but from changes in the haemoglobin to something like haematin, the pus frequently has a dirty brown colour. The suppuration may extend through the walls of the vein and lead to an abscess outside the vein; and the mesentery may thus contain an elongated abscess many times larger than the superior mesenteric vein. Thus, in some cases of pylephlebitis there is an abscess behind the pancreas.

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[1] Rolleston and Strangeways Pigg. *Journ. Path. and Bacteriol.*, 1898, v, 221.
[2] Bright, R. *Guy's Hosp. Rep.*, 1836, i, 632.
[3] Bristowe, J. S. *Trans. Path. Soc.*, 1858, ix, 285.

As a very rare accident in pylephlebitis Hodenpyl's[1] case may be referred to. A young man aged twenty-five died from rupture of the portal vein, the abdominal cavity being filled with pus. The pylephlebitis was due to streptococcic infection.

The extent of the vein affected may vary very greatly; a localised part of the portal vein or one of its branches may be found full of pus and shut off from the remainder of the vein by a partition of firm clot. Thus, when the trunk of the portal vein is suppurating the splenic vein is sometimes cut off from the infective process by a firm clot at its junction with the portal vein. Occasionally there are two foci of suppuration, one near the periphery, the other in the intrahepatic branches of the portal vein, while the intervening parts of the vein are healthy. In such cases the intrahepatic focus is due to emboli derived from the other focus of suppuration.

The *liver* is nearly always involved, either by an extension to it of the suppurative inflammation of the portal vein, or by secondary abscesses due to emboli from the portal vein. In very rare cases, of which I have examined one, the liver may be free from all signs of acute inflammation; in this case, however, suppurative inflammation had only recently supervened on thrombosis of the vein.

The liver is nearly always enlarged and may be double its natural weight. There is usually perihepatitis due to extension of inflammation to the capsule, which commonly leads to adhesions to the diaphragm. The surface of the organ is generally smooth, but abscesses may project somewhat from the surface and, if seen during an operation, may suggest multiple new-growth. Over these abscesses there may be a thin layer of recent lymph. The liver has a mottled appearance; the abscesses, or the areas where suppuration is about to take place, are of a palish-yellow colour. Before abscess formation occurs, coagulation necrosis of the liver cells may render parts of the liver firm and like nodules of secondary carcinoma. On section multitudes of small abscesses, varying in size from a millet-seed to a walnut, may be seen, due to the transverse section of the suppurating portal canals. By following the course of the portal canals the suppuration can be seen to spread out from the portal fissure into the liver like the branches of a tree. There is often dark-green staining of the portal canals from decomposition. One lobe, or only a

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[1] Hodenpyl. *Med. Rec.*, N.Y., 1898, liv, 133.

Bacillus coli is the
Commonest of the
various organisms
that may be found

limited part of a lobe, or again the whole liver, may shew abscesses. The left lobe escapes more often than the right. From the coalescence of a number of originally separate abscesses a large (areolar) abscess may be formed. In other instances the abscesses are so close to each other that a honeycombed condition of part of a lobe results.

The abscesses due to pylephlebitis may resemble at first sight some cases of suppurative cholangitis with abscesses in the liver. A careful examination of the bile-ducts and portal vein and their branches inside the liver should therefore always be made in any case of doubt. Similarly any difficulty in differentiating between tuberculous abscesses in connexion with the bile-ducts and pylephlebitis can, if necessary, be settled by microscopic examination. Multiple pyaemic abscesses, due to emboli conveyed by the hepatic artery, are small and hardly ever resemble pylephlebitis. The condition of multiple abscesses, due to embolism of the portal vein from some focus in its tributaries, only differs from pylephlebitis in the absence of phlebitis of the portal vein; in other respects the two conditions are practically identical.

Leakage or rupture of the small abscesses on the surface of the liver will set up peritonitis, either general or local, such as a subphrenic abscess. When the diaphragm is previously adherent to the liver, extension of infection may set up pleurisy, empyema, or suppuration in the lower lobe of the lung, usually on the right side. Abscesses on the under surface of the liver may give rise to perihepatic or subhepatic collections of pus.

Histology.—Microscopically the portal vein is filled with fibrin and leucocytes, its walls are infiltrated with small round cells, and may be destroyed. In the liver the portal spaces are similarly affected and small-celled infiltration extends between the lobules and around the intralobular branches of the vein. The liver cells at the periphery of the lobules are compressed and become elongated and look somewhat like oval connective-tissue cells; the small-celled infiltration spreads between the liver cells, which undergo degenerative changes. Micro-organisms are seen in the vessels and between the cells.

Bacteriology.—Streptococci, staphylococci, diplococci, and *Bacillus coli* in pure culture have been met with. In dysenteric cases the *Entamoeba histolytica* and other micro-organisms, and in cases after typhoid fever the *Bacillus typhosus*, have been found. Foulerton[1] has described streptotrichal pylephlebitis. Mixed infections are not uncommon.

In Norris's[2] case two anaerobic micro-organisms, a streptococcus resembling the *Micrococcus foetidus* and a new bacillus, were regarded as the cause of suppuration.

The Spleen.—There is usually some enlargement, which may be explained as due to the general infective or toxic condition. In cases in which the splenic vein is occupied by suppurating clot or in which the junction of that vein with the trunk of the portal vein is excluded, the spleen may be very large from venous obstruction. Abscesses are sometimes found in the spleen, and may either be part of suppurative pylephlebitis or its starting-point.

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[1] Foulerton. *System of Medicine* (Allbutt and Rolleston), 1906, ii, part i, 312.

[2] ~~Norris. *Journ. Med. Res.*, 1901–5, vi, 97.~~

Peritonitis is common; according to Bryant, it occurs in 50 per cent of the cases. It may be due to rupture of the pylephlebitic abscesses on the surface of the liver; it may be general or localised, as a subphrenic abscess usually on the right side. Peritonitis is also frequent around the primary lesion, such as appendicitis.

The pancreas is usually healthy; occasionally it contains abscesses, probably as a result of suppurative phlebitis of the splenic vein spreading into its pancreatic tributaries. An abscess behind the pancreas and around the portal vein sometimes results from suppurative pylephlebitis. An abscess due to suppurative pancreatitis may cause suppurative inflammation of the portal vein.

Pleura.—The right pleura is seldom healthy; there may be recent pleurisy, with or without a serous or purulent effusion. Inflammation readily spreads through the diaphragm from the liver. Rupture of a pyaemic abscess in the lung may set up an empyema or even a pyopneumothorax.

Lungs.—The right lung may shew collapse and hypostatic congestion of the base; abscesses with surrounding pneumonic consolidation may be due to direct extension through the diaphragm, or in rare instances to general pyaemia, some pylephlebitic abscesses having discharged into the hepatic veins.

Pylephlebitis with multiple small abscesses in the liver may give rise to general haemic infection and secondary abscesses in other organs. According to Langdon Brown, this occurs in 40 per cent of the cases.

In a case of pylephlebitis due to appendicitis, there were numerous small abscesses in the brain (Norman Moore[1]), and in Cassirer's[2] case the symptoms were those of a cerebral lesion, the cause—a fish-bone in the appendix—being only found at the necropsy.

Clinical Picture.—The *onset* of symptoms is more often sudden than gradual. In 42 cases analysed by Langdon Brown the onset was acute in 27, gradual in 15. It may be ushered in by rigors, but usually abdominal pain is the earliest symptom. The initial symptoms may be those of the disease—most often appendicitis—causing the pylephlebitis. Thus, pain, vomiting, and abdominal distension may be the first indications that anything is amiss. In a characteristic case there should be, first, evidence of the primary cause, such as appendicitis; secondly, ~~the development of~~ a pyaemic ~~septicaemic~~ state; and, thirdly, evidence that the liver is involved (Taylor[3]).

Course.—After the onset the patient passes into a state resembling pyaemia; rigors may be present at first, but usually disappear later. After a time evidence of hepatic disease, such as pain, enlargement, and

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[1] Norman Moore. *Trans. Path. Soc.*, Lond., 1882, xxxiii, 186.
[2] Cassirer. *Med. Press and Circ.*, 1901, cxxiii, 466.
[3] Taylor, F. *Guy's Hosp. Rep.*, 1902, lvi, 109.

∧| Haemorrhagic infarction has been described (Chabrot).

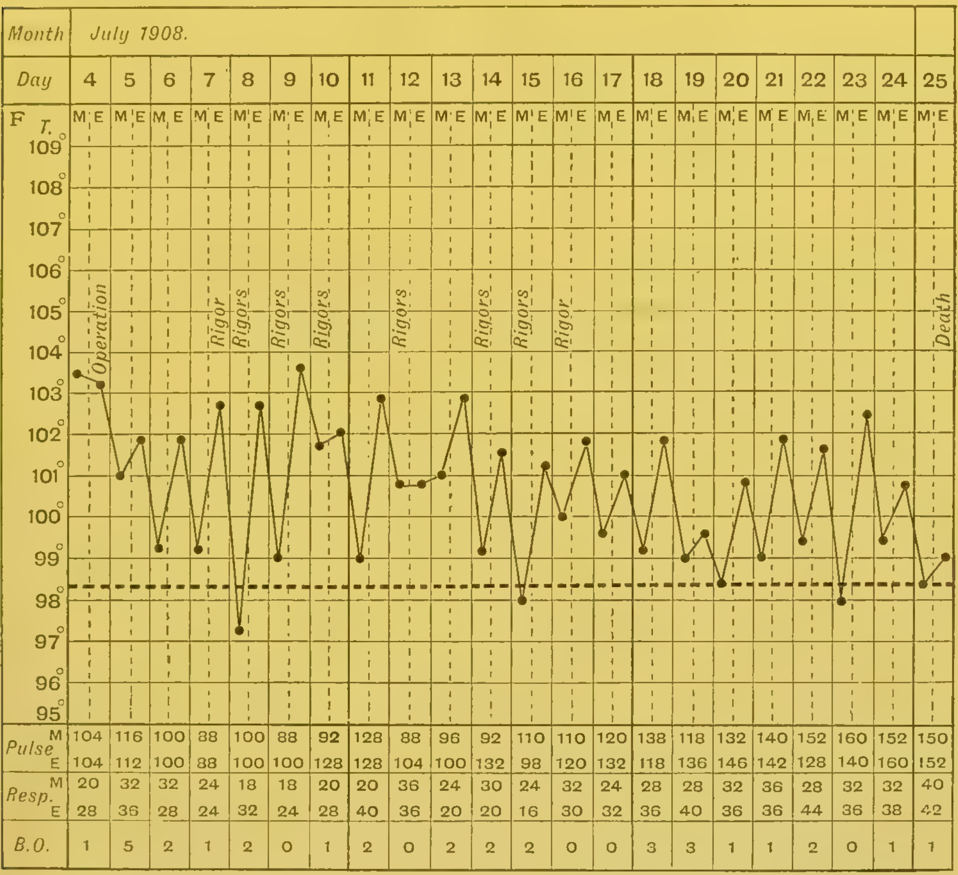
∧| , especially rigors (Thalhimer)

Chabrot. Semaine méd., Par., 1913, xxxiv, 61

Thalhimer, W. Arch. Surg., Chicago, 1924, viii, 658

tenderness, is forthcoming in the majority of cases; and signs suggesting empyema, pneumonia of the right lung, or peritonitis may appear. Gradually increasing weakness, often passing into coma, precedes death.

Duration.—There is considerable variation in the duration of the disease. The average of the cases collected by Langdon Brown[1] was forty-seven days, the shortest being three days. In an exceptional case of Goodhart's,[2] in which partial recovery occurred, the duration was two hundred and ninety-six days.



Day		4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
Pulse	M	104	116	100	88	100	88	92	128	88	96	92	110	110	120	138	118	132	140	152	160	152	150
	E	104	112	100	88	100	100	128	128	104	100	132	98	120	132	118	136	146	142	128	140	160	152
Resp.	M	20	32	32	24	18	18	20	20	36	24	30	24	32	24	28	28	32	36	28	32	32	40
	E	28	36	28	24	32	24	28	40	36	20	20	16	30	32	36	40	36	36	44	36	38	42
B.O.		1	5	2	1	2	0	1	2	0	2	2	2	0	0	3	3	1	1	2	0	1	1

FIG. 12.—Temperature chart of man, aged 22, who was attacked with appendicitis on June 25, had a retrocaecal abscess opened on July 4, and died with pylephlebitis on July 25.

Signs.—The patient has an abdominal facies, looks sallow, ill, and anxious, and has a raised temperature. The type of fever varies very widely; it may be continuous, intermittent, or remittent, and towards the close, subnormal. The pulse is soft, compressible, and rapid (100 to 140). The respirations are quickened. When the disease has lasted some time wasting and prostration appear. Signs of portal obstruction, which are the rule in pylethrombosis, are exceptional in suppurative pylephlebitis.

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[1] Brown, Langdon. *St. Barth. Hosp. Rep.*, 1901, xxxvii, 128.

[2] Goodhart. *Trans. Path. Soc.*, Lond., 1882, xxxii, 137.

Rigors are seen in a majority of the cases.

In 42 cases Langdon Brown found them in 29, or 69 per cent; in Bryant's series they were present in half the cases.

The rigors occur during the earlier part of the illness and tend to disappear in the later stages. They are followed by profuse sweating, and are uncontrolled by quinine.

Jaundice occurs in less than half the cases and is often quite slight, transient, or only noted late in the course of the disease, and can be explained as due to the general toxaemia. In some cases it is marked and may be due to cholangitis, concomitant obstruction of the bile-duct by calculi, or to pressure of an abscess on the common bile-duct.

In 44 cases, tabulated by Langdon Brown, there was jaundice in 19; in 4 of these it was very slight or transient, while in 5 it was an initial sign.

The *liver* can be felt to be enlarged in more than half the cases; Frerichs says in 75 per cent, Bryant in 60 per cent, and Langdon Brown, who collected a larger number of cases, in 57 per cent of the cases. The enlargement is usually uniform, but in a few instances elevations, due to projecting abscesses, are distinctly palpable. The enlarged liver may reach down to the umbilicus and may lead to upward displacement of the diaphragm, and thus to collapse of the lower lobe of the right lung and even to displacement of the apex of the heart. A friction rub from inflammation of its peritoneal surface may be heard over the liver. Pain and tenderness in the hepatic region are generally met with, but they may be absent. Local tenderness, which is sometimes present when the liver cannot be felt to be enlarged, is more valuable as evidence of hepatic suppuration than pain referred to the right hypochondrium.

Enlargement of the spleen is not common; in 53 cases it was noted in 11 (Langdon Brown). When there is very considerable enlargement, haematemesis should be expected; because the size of the spleen may be due to obstruction of the splenic vein, which would also lead to engorgement of the cardiac end of the stomach by interfering with the return of blood through the vasa brevia veins.

Vomiting is often met with early in the course of the disease. It was present in 24 out of 42 cases collected by Langdon Brown, being an initial symptom in 11. It is not so much a sign of pylephlebitis as of the disease, such as appendicitis, responsible for it.

In rare instances a *gastric ulcer* may be secondary to pylephlebitis and even go on to perforation.

In a man aged fifty-four examined by me in 1897, with suppurative pylephlebitis secondary to appendicitis, there was perforative peritonitis due to rupture of a gastric ulcer, which, in its turn, was evidently due to thrombosis of a gastric vein.

Diarrhoea occurs in about half the cases. Blood is occasionally seen in the stools. *Constipation* is rarely noticed. *Hiccup* may be troublesome.

When the hepatic veins become infected micro-organisms pass into the general circulation.

The great majority of the cases are secondary to an unrecognized appendix abscess, such as is especially likely to occur in the pelvis; hence a rectal examination should be undertaken.

Tympanitic distension of the abdomen may occur from peritonitis and will obscure enlargement of the liver and spleen.

Ascites is rare. It was met with in 6 out of 53 cases (Langdon Brown[1]). It is more likely to occur in cases that last a considerable time, and when suppuration supervenes on thrombosis of the portal vein. Although clinically ascites is seldom seen, there may be a small amount of ascitic effusion, due to local acute peritonitis over the abscesses in the liver.

Thoracic Signs.—A friction rub may be audible over the right side of the chest from extension of inflammation through the diaphragm to the pleura. The pressure exerted by the enlarged liver may cause bulging of the chest wall and collapse of the lung, imitating pleurisy with effusion. In some cases there may actually be a purulent effusion into the pleura or the lung may contain abscesses. Such cases may be diagnosed as empyema or septic pneumonia.

Blood.—There is some secondary anaemia, especially in prolonged cases. Leucocytosis, which would naturally be expected, was present in 3 out of 4 cases referred to by Langdon Brown; and in 4 cases, associated with appendicitis, French[2] found marked leucocytosis (24,000) in one and in the others counts of 18,000, 12,000, and 10,500. In 15 cases, investigated by Libman,[3] blood-cultures were sterile in 14; in one accompanied by calculous cholangitis the blood contained pneumococci.

Urine.—Albuminuria is sometimes found and is due to toxic absorption. When there is jaundice bile-pigment will be found in the urine. Indicanuria may be present, and excessive urobilin has been observed (Goodfellow[4]).

Termination.—Towards the end the patient passes into a condition of stupor. Death may occur from peritonitis, from coma and increasing weakness, suddenly, or in rare instances from collapse after haematemesis or melaena.

Diagnosis.—A septicaemic or pyaemic state especially rigors with evidence that the liver is affected, ~~in a patient who has had~~ appendicitis or some other intra-abdominal condition known to cause pylephlebitis, forms the broad outline of the disease. Enlargement and tenderness of the liver, with pain, fever, and a pyaemic state, not due to any other cause, should suggest suppurative pylephlebitis. Pyaemia from bone and ear disease and infective endocarditis must be excluded. But a diagnosis is not always possible, and since hepatic enlargement is absent or is not detected in a certain proportion of cases of pylephlebitis, there may be nothing to direct attention to the liver. The difficulty of diagnosis is shewn by the fact that in 20 cases examined after death in Guy's Hospital a correct diagnosis during life was only arrived at in two instances during life (Bryant[5]).

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[1] Brown, Langdon. *Brit. Med. Journ.*, 1905, ii, 1393.
[2] French, H. S. *Med.-Chir. Trans.*, 1904, lxxxvii, 485.
[3] Libman. *Am. Journ. Med. Sc.*, Phila., 1908, cxxxvi, 548.
[4] Goodfellow. *Med. Chron.*, 1903, xxxvii, 288.
[5] Bryant, J. H. *Guy's Hosp. Rep.*, 1900, liv, 77.

Differential Diagnosis.—In *tropical abscess* there is not such rapid emaciation and prostration as in pylephlebitis, fluctuation may be felt, a history of past dysentery rather than of appendicitis should be forthcoming, and the spleen is not enlarged, as it may be in pylephlebitis. In many cases it is impossible to be certain whether there is a single abscess deeply situated in the liver or pylephlebitis with multiple hepatic abscesses. As mentioned above, the history of pre-existing dysentery is generally in favour of a single abscess, but it must be remembered that dysentery may be followed by multiple abscesses in the liver like those of pylephlebitis; this was the case in the dysentery seen in the South African War of 1899–1902 (*vide* p. 118).

In *hepatic suppuration* secondary to appendicitis multiple foci of suppuration are the rule, but in exceptional instances a large areolar abscess, probably due to the union of originally independent abscesses, results. In a given case with hepatic enlargement, fever, rigors, and pain, the diagnosis between multiple abscesses, pylephlebitis, and a single abscess is very difficult, and probably the patient should be given the benefit of the doubt and abdominal exploration be undertaken. Oedema of the chest wall or definite fluctuation justifies a diagnosis of a large and probably single abscess.

In *suppurative cholangitis* jaundice is commoner, appears earlier, and is more prominent than in pylephlebitis. Splenic enlargement may occur in both, but is more likely to be marked in pylephlebitis. A history of cholelithiasis and biliary colic is in favour of suppurative cholangitis. There may, however, be very slight or no jaundice in suppurative cholangitis and no history of gall-stones, and the diagnosis from pylephlebitis may be impossible, as in case recorded on p. 675.

Syphilitic disease of the liver with fever (*vide* p. 364) may closely simulate suppurative pylephlebitis (Edwards[1]).

Subphrenic abscess may be due to rupture of a pylephlebitic abscess, and may be opened or drained, and the underlying condition be unsuspected unless the liver is much enlarged. In cases of pylephlebitis supervening after appendicitis, but without any manifest hepatic enlargement, there may be a very close resemblance to subphrenic abscess in the right kidney pouch.

Enteric Fever.—In enteric fever the agglutination (Widal's) reaction with typhoid bacilli should be present, while rigors and jaundice, though not unknown, are decidedly rare. A positive agglutination reaction may occur in pylephlebitis when the patient has had enteric fever some time previously.

In a woman aged twenty-eight, who died of pylephlebitis under Dr. F. Taylor's[2] care, there was a positive agglutination reaction with typhoid bacilli although she had not had enteric fever.

Pylephlebitis may closely resemble enteric fever, especially when there

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[1] Edwards. *Amer. Journ. Med. Sc.*, Phila., 1910, cxl, 527.

[2] Taylor, F. *Guy's Hosp. Rep.*, 1902, lvi, 109.

HELLSTRÖM. Beitr. z. klin. Chir., Tübingen, 1912,
LXXX, 51

is blood in the stools. Bryant lays stress on the difference between the pulse, temperature, and respiration rates; the pulse rate is low (80) in enteric, high (140) in pylephlebitis.

In *infective endocarditis* murmurs are occasionally absent and the aspect of the case is one of pyaemia of obscure origin; in such cases, when accompanied by enlargement of the liver and spleen, the resemblance to pylephlebitis may be considerable. In such cases the discovery of micro-organisms in the blood by means of blood-cultures points to infective endocarditis. A negative result is in favour of pylephlebitis (Libman).

In chronic cases of *malarial infection* the enlarged and sometimes very tender liver and the raised temperature associated with shivering and sweating may lead to considerable difficulty in diagnosis. Malaria should be recognised by the presence of the parasite in the blood and improvement under quinine; further, the patient's condition is not so grave as in pylephlebitis.

Intra-thoracic conditions, such as pleural effusion, empyema, pulmonary abscess or pneumonia, may be diagnosed as the sole condition, from physical signs depending on the spread of inflammation from the liver in pylephlebitis.

Malignant disease of the liver may imitate pylephlebitis when fever and rigors accompany rapid increase of the malignant growth. A case of secondary endothelioma of the liver resembling pylephlebitis is described on p. 522.

In *acute cirrhosis* the enlarged and tender liver and the presence of fever may imitate pylephlebitis (Carrington[1]).

Prognosis.—Since diagnosis is difficult, more cases are recognised on the post-mortem table than during life, and grave doubt must exist as to the nature of cases regarded as suppurative pylephlebitis which have recovered without laparotomy and examination of the liver. It is in the highest degree improbable that widespread suppuration of the portal vein would be followed by recovery, and the general consensus of opinion is that the disease is uniformly fatal. Conceivably this verdict may require some modification in the light of more frequent observations of the state of the liver as seen in the course of exploratory laparotomies. In isolated cases recovery has occurred in which multiple abscesses were thought to exist, but some question must arise if suppurative inflammation of the portal vein was present.

Quénu and Mathieu[2] collected 13 cases of recovery after operation on liver abscesses due to appendicitis; Treves[3], ~~and~~ West[4] recorded ~~two~~ similar cases. HELLSTRÖ Cases presenting appearances at the necropsies of healed suppurative pylephlebitis have been recorded by Goodhart[5] and Moschcowitz.[6]

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[1] Carrington. *Guy's Hosp. Rep.*, 1883–4, xlii, 337.
[2] Quénu et Mathieu. *Rev. de chir.*, Par., 1911, xliv, 519.
[3] Treves. Allbutt's *System*, 1897, iii, 927; *Lancet*, 1894, i, 662.
[4] West, S. *Trans. Clin. Soc.*, 1884, xvii, 126.
[5] Goodhart. *Trans. Path. Soc.*, 1882, xxxii, 137.
[6] Moschcowitz. *Ann. Surg.*, 1911, liii, 551.

When the disease is recognised, **treatment** is usually confined to relieving pain and other symptoms. Langdon Brown and Gerster[1] employed antistreptococcic serum without any good results. A trial of such a serum or vaccine should be made on the chance that the infection of the portal vein is a pure streptococcic one, but in most cases there is a mixed infection. Treatment with polyvalent serums, viz. from animals immune against various strains of micro-organisms of the same and of different species (streptococci, *Bacillus coli*), should be employed. Since it is difficult or impossible to be sure that symptoms of pyaemia with hepatic enlargement after appendicitis are not due to a single abscess, operative interference gives the patient a chance of recovery. Further, it is possible that success might follow the opening and draining of several abscesses. When operation is undertaken, the liver should be freely exposed. Aspiration through the abdominal wall is not only fallacious, but dangerous.

Prophylactic treatment is most important, and consists in the early and radical removal of conditions, such as appendicitis, likely to induce suppurative pylephlebitis. During such operations removal of any suppurating veins in the immediate neighbourhood is an important step in preventing the spread of infection from the intestinal branches to the trunk of the portal vein. This was carried out in two cases of appendicitis which recovered (Gerster). It is also important not to disturb the parts unnecessarily, so as to avoid detaching a thrombus and producing embolism of the intrahepatic branches of the portal vein.

OTHER AFFECTIONS OF THE PORTAL VEIN

Embolism; Endophlebitis; Calcification; Parasites; Angioma.

Embolism of the intrahepatic branches of the portal vein is frequent in suppurative inflammation of the intestinal tributaries of the portal vein and accounts for multiple abscesses in the liver after appendicitis, etc. In most cases the emboli are small and do not themselves attract attention. Similarly embolism plays an important part in the production of secondary growths in the liver in carcinoma of the stomach and intestine. Sometimes large embolic masses of clot may be found in the main intrahepatic branches of the portal vein.

In 17 cases of infarcts in the liver Chiari[2] found that 15 were due to embolism of the portal vein, the emboli being derived from thrombosis in its peripheral tributaries, such as the haemorrhoidal, gastric, splenic and mesenteric veins.

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[1] Gerster. *Med. Rec.*, N.Y., 1903, lxiii, 1005.

[2] Chiari. *Ztschr. f. Heilk.*, 1898, xix, 475.

Recovery has occurred ~~in a case that [illegible]~~ after intravenous injections of eusol (Barlow).

Ligature of the trunk of the portal vein

Barlow Lancet, 1919, i, 844.

Endophlebitis.—Acute inflammation of the portal vein is practically the same as pylephlebitis. *Chronic endophlebitis* of the portal vein is not uncommonly associated with hepatic cirrhosis, and may be explained as the result of increased blood-pressure in the vein, combined perhaps with degenerative and hyperplastic changes due to the action of toxic bodies. There is a certain amount of chronic endophlebitis of the portal vein in long-standing backward pressure from tricuspid regurgitation, in mitral disease, and in obstructive pulmonary diseases, such as emphysema. According to Borrmann,[1] syphilitic disease of the liver may extend into the portal vein and set up endophlebitis of its walls. But in a certain number of cases the endophlebitis is primary and the liver merely shews a secondary atrophy. The cause of the chronic inflammatory change in the vein is somewhat obscure, and has been referred to various factors. Inflammation may also spread into the vein walls from chronic peritonitis. In splenic anaemia endothelial hyperplasia may spread from the blood sinuses in the spleen into the splenic and portal veins; the terminal cirrhosis of the liver in splenic anaemia (Banti's disease) has been thus explained. Chronic endophlebitis may lead to calcification and to thrombosis; the latter result is of practical importance.

Calcification is a sequel of long-standing endophlebitis and is analogous to secondary calcification in endarteritis. It is probably much commoner than is usually thought; it is not infrequent in chronic splenic anaemia and Banti's disease. I have seen it microscopically in cases in which there was not sufficient infiltration to attract attention in the post-mortem room. It has long been recognised; Frerichs[2] gives references to "ossification" or calcification of the portal vein. The calcareous plates or spicules may project into the lumen of the portal vein and set up thrombosis. But calcification may be very extensive without any trace of thrombosis, and it must not be assumed, as Lancereaux[3] did, that calcification of the wall of the portal vein is merely the result of thrombosis.

A high grade of chronic endophlebitis with calcification of the splenic and portal veins occurred in a man aged fifty-three who died in St. George's Hospital after an operation for hydrocele. The splenic vein was greatly dilated and the splenic artery was very calcareous and had three aneurysms on it. The spleen was much enlarged (42 ounces) and fibrotic. The liver (37 ounces) was scarred, and to the naked eye suggested syphilis, but microscopically it shewed multilobular cirrhosis of old standing and no evidence of syphilis. The colon shewed extensive ulceration, possibly of vascular origin, but there was no thrombosis in the mesenteric arteries or in any part of the portal vein or its branches.[4]

Parasites in the Portal Vein.—*Schistosomum haematobium*, endemic in Egypt, Natal, Mauritius, and Syria, may be found in the trunk of the portal vein or in its haemorrhoidal tributaries, while the walls may contain the ova. The liver may shew slight cirrhosis,[1] but there are no symptoms associated with the presence of the worms or their ova in the portal vein. The liver fluke (*Fasciola hepatica*) was described in a case quoted by Budd[2] as being found in the portal vein, but it is conceivable that a dilated bile-duct was mistaken for the vein.

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[1] Borrmann. *Deutsches Arch. f. klin. Med.*, 1897, lix, 283.

[2] Frerichs. *Diseases of the Liver*, 1861, ii, 402. Transl. New Sydenham Soc.

[3] Lancereaux. *Traité des maladies du foie et du pancréas*, 1899, p. 571.

[4] Trevor, R. S. *Trans. Path. Soc.*, Lond., 1903, liv, 302.

A **haemangioma** may arise in its wall and occlude the portal vein.[3]

MORBID CONDITIONS OF LYMPHATIC VESSELS AND GLANDS

Morbid Conditions of the Lymphatic Vessels.—Comparatively little is known on this subject, and primary affections of the lymphatics of the liver are not recognised. The two main sets of lymphatics, (*a*) those which run along the portal spaces, and (*b*) those which supply the capsule or are superficial, may be separately affected. The lymphatics frequently suffer when the portal spaces are affected by morbid processes such as inflammation and tuberculosis, as shewn by the enlargement of the lymphatic glands in the portal fissure. Inflammation may thus spread from the liver to the pancreas. If from acute inflammation the lymphatics are obstructed, resolution and absorption of inflammatory products in the liver are prevented and cirrhosis may result (Oertel[4]). In chronic perihepatitis the superficial lymphatics are exclusively affected.

Pericholangitis is very closely connected with lymphangitis of the portal spaces. Thus, the glands in the portal fissure are enlarged in hypertrophic biliary cirrhosis, tuberculous cavities in the liver, cholangitis, and pylephlebitis, all of which are inflammatory lesions involving the tissues and the portal canals around the bile-duct and portal vein. In a case of splenic anaemia of the Gaucher type endothelial proliferation in the lymphatic vessels of the portal spaces is figured by Bovaird.[5]

New-growth may sometimes be seen working its way into the liver, against the lymph-stream, along the lymphatics of the portal fissure; more commonly the glands in the portal fissure become infected secondarily to a growth in the liver, the infecting cells travelling in the normal direction along the lymphatic vessels. Distension of the lymphatic vessels in the portal spaces is occasionally due to obstruction; it has also occurred from torsion of the bile-duct in hepatoptosis (*vide* p. 32). In diabetic lipaemia I have seen the lymphatics of the portal spaces graphically mapped out by the contained fat. Maresch[6] described

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[1] Symmers. *Journ. Path. and Bacteriol.*, 1904, ix, 237.

[2] Budd. *Diseases of the Liver*, p. 485, 1852, ed. ii.

[3] Pick, L., *Virchows Arch.*, 1909, cxcvii, 490; Emmerich, *Frankfurter Ztschr. f. Path.*, 1912, x, 362.

[4] Oertel, H. *Arch. Int. Med.*, Chicago, 1908, i, 385.

[5] Bovaird. *Am. Journ. Med. Sc.*, Phila., 1900, cxx, 391.

[6] Maresch. *Ztschr. f. Heilk.*, 1903, xxiv, 39.

Look up:
[illegible]
Harvey lecture [illegible]?

a pedunculated lymphangioma removed from the right lobe of the liver of a girl aged five years.

The Lymphatic Glands in the Portal Fissure.—Any enlargement of these glands is important inasmuch as pressure may thus be exerted on the bile-ducts and jaundice set up. It has been suggested that the jaundice occasionally seen in the roseolous stage of syphilis may be due to swelling of the glands in the portal fissure. Enlargement of the portal glands may occur in lardaceous disease and leukaemia, but cannot be credited with producing jaundice or ascites by compression of the bile-duct or portal vein.

Enlargement of the portal lymphatic glands may be due to various conditions, chiefly inflammatory, inside the liver, such as abscess, pylephlebitis, suppurative cholangitis, tuberculosis, hypertrophic biliary cirrhosis, and primary carcinoma. As already mentioned, new-growth may extend into the portal fissure along the lymphatic vessels against the flow of lymph, and occasionally infiltration of the portal lymphatic glands may be secondary to carcinoma in the peritoneal cavity, and may give rise to jaundice.

CHRONIC VENOUS ENGORGEMENT OF THE LIVER

Synonyms: Nutmeg Liver, Cardiac Liver, Cyanotic Atrophy,
Hepatic Asystole.

THE term red atrophy has been applied to the small nutmeg liver seen in cases of long-standing backward pressure, but unfortunately it may lead to confusion with an entirely different condition, viz. the red atrophy seen in acute (yellow) atrophy. An old and now forgotten name for the condition of the liver was hypertrophy of the white substance.[1] Hanot[2] suggested the term hepatic asystole for those cases in which the hepatic symptoms are more prominent than those of the primary cardiac disease.

Etiology.—Chronic venous engorgement of the liver is practically always secondary to obstructive heart or lung disease. *Cardiac Lesions.*—Mitral obstruction or regurgitation, and some diseases of the myocardium, such as alcoholic dilatation, give rise to backward pressure, tricuspid regurgitation, and chronic hepatic engorgement. Tricuspid stenosis, which is practically always secondary to mitral stenosis, causes a high grade of hepatic engorgement. Mitral stenosis is the most effective cause of chronic venous engorgement of the liver. Acute dilatation of the heart, as in pneumonia or diphtheria, produces an engorged liver.

Lung Lesions.—Obstruction to the passage of blood through the pulmonary artery and its branches in the lungs first causes hypertrophy of the right ventricle, but eventually this compensation becomes strained, the right ventricle dilates, and tricuspid incompetence results. This train of events most often follows chronic bronchitis and emphysema, the pneumoconioses, and sometimes chronic interstitial pneumonia. It is remarkable that chronic venous engorgement is not more frequent in ordinary pulmonary tuberculosis. In my experience it is rare in chronic pulmonary tuberculosis, but Gilbert and Weil,[1] in a study of 25 livers, found that in 6 the naked-eye appearances were those of a typical nutmeg liver, and that in 5 more the microscopical appearances were undoubted. Possibly the cases in which it occurs are complicated by alcoholic dilatation of the heart.

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[1] Compare Hope. *Principles and Illustrations of Morbid Anatomy*, 1834, p. 102.

[2] Hanot. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1895, xii, 409.

Other Factors.—A tumour or aneurysm might press on the small segment of the inferior vena cava between the entrance of the hepatic veins and its termination in the right auricle; but it is extremely rare for an intra-thoracic growth to obstruct the inferior cava. I have seen nodules of secondary growth on the diaphragm in close contact with the inferior cava. A high grade of chronic venous engorgement of the liver is produced by occlusion of the hepatic veins (*vide* p. 48); but in a case of obstruction of the inferior vena cava involving their orifices the liver was cirrhotic rather than nutmeggy (Osler[2]).

Inside the liver the pressure of malignant growths or hydatid cysts may cause areas of chronic venous engorgement. It is possible that marked displacement of the heart in pleural or pericardial effusion, by producing kinking of the inferior vena cava, might lead to venous stasis in the hepatic veins of the liver as well as in the other tributaries of the inferior cava. It has been suggested that pericardial adhesions and chronic mediastinitis may lead to torsion or compression of the inferior vena cava and so to chronic venous engorgement. It has been thought that any pre-existing morbid condition of the liver, such as may result from alcoholism, biliary calculi, or malaria, renders the organ more prone to suffer from the effects of backward pressure in any given case of tricuspid incompetence.

Morbid Anatomy.—Adhesions between the upper surface of the liver and the diaphragm are common. They are the legacies of past attacks of acute perihepatitis which may be due to the spread of inflammation through the diaphragm from acute pleurisy or pericarditis. The upper surface of the right and left lobes often shews a depression corresponding to a hypertrophied and dilated heart; and the peritoneum in this area is often thickened and opaque as the result of increased and long-continued friction. In universal chronic perihepatitis or "iced liver" (*vide* p. 167) there is usually chronic venous engorgement; the stasis is not the cause of the capsular change but is due to cardiac failure, adherent pericardium, or possibly to narrowing of the orifices of the hepatic veins by the thickened capsule.

Size.—After death the liver is sometimes larger than natural, but

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[1] Gilbert et Weil. *Arch. de méd. expér. et d'anat. path.*, 1902, xiv, 729.

[2] Osler. *Journ. Anat. and Physiol.*, 1879, xiii, 291.

is smaller than in life. During life the liver is distensible and may vary in size very considerably within short periods as the result of alterations in the amount of backward pressure. After death, and the onset of rigor mortis, coagulation of the protoplasm of the liver cells gives a firmness to the organ which is not present in life.

Sir Lauder Brunton[1] found that if artificial circulation be kept up through the portal vein of an animal just killed, the liver will enlarge and diminish in response to variations in the pressure exerted, almost as if it were a sponge, and much in the same way as it does in cases of pulsating livers.

In the later stages the liver becomes smaller; this is most probably due to atrophy of the liver cells and not to fibrosis. The specific gravity of the liver is increased (Olsho[2]). The liver is of a darkish purple colour externally and is rather firmer than natural. Dilated veins may be seen on the surface, and no doubt their prominence is largely due to atrophy of the surrounding liver tissue. As a rule, the surface shews depressed lines of atrophy, and may thus become slightly uneven, suggesting to the naked eye early cirrhosis. The characteristic mottling is generally not well seen externally. In long-standing ascites, the serous covering of the liver is thickened or opaque.

On section the organ contains an unusual amount of blood and presents the characteristic "nutmeggy" appearance, which depends on the fact that the sublobular and intralobular veins are dilated and appear as dark purple spots or streaks according as they are cut transversely or longitudinally, while the other parts of the lobules, being stained with bile or infiltrated with fat, are paler than natural. This contrast between the dark and the pale areas resembles the section of a nutmeg. In a typical case the centre of the lobule appears as a dark spot; this is surrounded by a whitish-yellow margin due to fatty and slightly bile-stained liver cells. In other cases there is a coarse nutmeg appearance. Some white areas, though to the naked eye resembling fatty change, are nodules of regenerating liver cells without any fatty change or venous engorgement. The hepatic veins are dilated and their walls thicker and more opaque than natural; this phlebosclerosis is accompanied by atrophy and condensation of the hepatic tissue immediately around the vein walls.

Opponet,[3] in a thesis on hepatic asystole, puts forward the view that the incidence of hepatic manifestations in a case of heart disease is determined by a peculiar arrangement of the hepatic veins at their entry into the inferior vena cava; this anatomical disposition he regards as congenital and as fairly common. It seems, however, more natural to regard the size and manner of the openings of the hepatic veins into the inferior vena cava as the results of backward pressure.

In backward pressure in the inferior vena cava the main stress usually falls on the legs; occasionally, however, the renal veins are chiefly involved, as shewn by persistent albuminuria without any corresponding nephritis on histological examination, whilst in cases of hepatic asystole the hepatic veins suffer chiefly.

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[1] Lauder Brunton in Murchison's *Diseases of the Liver*, p. 134, 3rd ed., 1885.

[2] Olsho. *Arch. Int. Med.*, Chicago, 1909, ii, 171.

[3] Opponet. *Thèse de Paris*, 1895.

Occasionally a liver shewing chronic venous engorgement is also distinctly cirrhotic. Thus, a patient with cirrhosis may die with alcoholic dilatation of the heart or with backward pressure from cardiac failure due to other causes. Chronic venous engorgement and cirrhosis are both common and sometimes coincide, but chronic venous engorgement alone does not cause real cirrhosis. This is my firm conviction from an examination of a large number of livers microscopically.

The Question of "Cardiac Cirrhosis."—For a full account of the question whether or not chronic venous engorgement *per se* leads to cirrhosis of the liver the reader should consult Piery's thesis.[1] In 1840 Becquerel[2] stated that chronic venous engorgement was the cause of half the cases of cirrhosis examined by him; his view was supported by Rokitansky,[3] Henle,[4] and others. Possibly, as suggested by Budd, cirrhotic and nutmeg livers were not always differentiated from each other at this time. In 1845 Budd[5] and in 1848 Handfield Jones, who definitely recognised that these two conditions were distinct, stated that chronic venous engorgement did not give rise to cirrhosis.

There has been some discussion as to the situation of the fibrosis in cardiac cirrhosis. Rokitansky and Sabourin[6] described it as centrilobular, whilst English observers—Handfield Jones,[7] Green,[8] and Legg[9]—considered that it was perilobular but not genuine multilobular cirrhosis. Numerous observations prove that chronic venous engorgement of the liver may exist for prolonged periods without producing any fibrosis at all, or only a slight fibrosis, as seen microscopically, which can be satisfactorily accounted for by atrophy of the liver cells and secondary fibrous replacement. The firm appearance of many nutmeg livers often suggests a fibrosis which can only be disproved by microscopic examination. The belief that chronic venous engorgement induces cirrhosis is probably based on naked-eye observation uncorrected by a further microscopic examination. Parmentier[10] experimentally produced chronic venous engorgement of the liver in animals by setting up tricuspid regurgitation, and found that fibrosis did not follow.

In the rather exceptional instances presenting more fibrosis, some

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[1] Piery. *Arch. gén. de méd.*, Par., 1900, clxxxvi, 582.
[2] Becquerel. *Ibid.*, 1840, 3. s., vii, 397.
[3] Rokitansky. *Handb. der path. Anat.*, 1842, iii, 347.
[4] Henle. *Ztschr. f. inn. Med.*, 1844.
[5] Budd. *Diseases of the Liver*, 1845, p. 118.
[6] Sabourin. *Rev. de méd.*, 1883, iii, 523.
[7] Handfield Jones. *Med. Gaz.*, 1848, vii, 1033.
[8] Green. *Introduction to Pathology*, 1871, p. 244.
[9] Legg, Wickham. *Med.-Chir. Trans.*, 1875, lviii, 345.
[10] Parmentier. *Études cliniques et anatomo-pathologiques sur la foie cardiaque*, p. 29, Paris, 1890.

other factor, such as prolonged gastro-intestinal catarrh or considerable alcoholic stimulation during the course of chronic valvular disease, may be invoked. In chronic venous engorgement the resistance of the liver is necessarily reduced, and the organ is therefore more likely to be affected by poisons than in health. It would thus be reasonable to expect to find cirrhosis secondary, not to chronic venous engorgement pure and simple, but to factors such as alcoholism, auto-intoxication from intestinal fermentation (dyspeptic or Budd's cirrhosis), or bacterial infection. Piery believes that tuberculosis and acute rheumatism may also be responsible for hepatic cirrhosis in patients with heart disease.

Salaman[1] describes the cirrhotic nutmeg liver as due to repeated attacks of severe cardiac failure which have led to destruction of hepatic cells and subsequent scarring, the ordinary nutmeg liver being due to steady backward pressure. The livers are small, the capsule thickened and irregular, and on section are generally pale and shew a confused "nutmeggy" aspect.

Still, with all these different ways in which cirrhosis may become implanted on a nutmeg liver, the association of well-marked cirrhosis with chronic venous engorgement is infrequent.

Comparison of Chronic Venous Engorgement in the Liver and Lung.—Both these organs have a double blood-supply, the portal vein and the hepatic artery in the one case, and the pulmonary and bronchial arteries in the other. While pulmonary apoplexies are frequent, the structure of the liver does not allow extensive extravasation of blood into the bile-ducts, for haemorrhage into the bile-capillaries and ducts would constitute the exact counterpart of pulmonary apoplexies. Comparatively large areas of haemorrhage into the liver substance occur, however, in chronic venous engorgement. Bonome[2] described haemorrhagic infarcts in nutmeg liver.

Microscopic Appearances.—The conditions vary in different stages of the disease and in different parts of the same liver. The intralobular veins and their capillaries (blood-spaces or sinusoids of Minot) are first dilated. This dilatation is at first limited to the central zone, but in the more advanced stages spreads into the intermediate zone. Eventually large areas composed of blood corpuscles with some fibrin result.

In 35 cases of advanced chronic venous engorgement Hart[3] found capillary thrombosis near the centre of the lobules constantly.

The liver cells are compressed, present varying degrees of distortion, and may undergo necrosis and so leave spaces filled with blood which resemble, but are not, sinusoids; this extreme change is said not to be due to uncomplicated chronic engorgement, but to depend on toxins (Mallory,[4]

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[1] Salaman. *Lancet*, 1907, i, 4.

[2] Bonome. *Arch. di biol.*, Firenze, 1899, liii, 319.

[3] Hart. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1904, xxxv, 303.

[4] Mallory. *Journ. Med. Res.*, 1901 [illegible], vi, 264.

Frothingham [1]). Oertel,[2] however, regards it as due to cytolytic ferments derived from the atrophied liver cells. The liver cells contain an iron-free pigment—haematoidin—derived from haemoglobin, which differs from the iron-containing pigment (haemosiderin) deposited in the peripheral zone of the lobule in pernicious anaemia in not striking a blue colour with dilute hydrochloric acid and ferrocyanide of potassium (Perl's test).

The liver cells tend to shew varying degrees of fatty change, and in long-standing cases may contain large globules of fat. This fatty change is not, however, universally met with. It is usually stated that this fatty change is mainly in the peripheral zone of the hepatic lobule; Salaman, however, insists that it is situated in the central zone. The cells near the area of engorgement sometimes shew coagulation-necrosis (Opie [3]). In some parts of the liver the central zones may become compressed by compensatory hyperplasia of the persistent peripheral zones of adjacent lobules which may coalesce (Géraudel [4]); the appearances may thus be confusing, as the portal canals appear to be in the centre of the lobules. Compensatory hyperplasia of the liver cells giving rise to the formation of regeneration nodules, which are free from chronic venous engorgement, is common. This process, first described by Letulle [5] and by Jacobi,[6] resembles that seen in nodular cirrhosis.

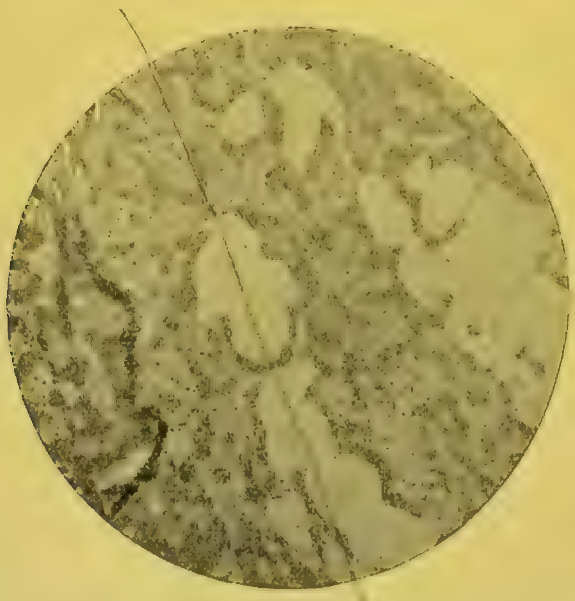

FIG. 13.—Photomicrograph of chronic venous engorgement. Shews extravasated blood and distorted liver cells (Dr. S. G. Penny).

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[1] Frothingham. *Arch. Int. Med.*, Chicago, 1910, v, 1.

[2] Oertel. *Ibid.*, 1910, vi, 293.

[3] Opie. *Trans. Assoc. Amer. Phys.*, 1904, xix, 132.

[4] Géraudel. *Arch. de méd. expér. et d'anat. path.*, 1906, xviii, 514.

[5] Letulle. *Anatomie pathologique*, 1897, i.

[6] Jacobi, A. *Trans. Assoc. Am. Phys.*, 1897, xii, 500.

I have seen two well-marked examples of this condition, which at first sight resembles multiple caseous tubercles.

The fibrous trabeculae contract and become more prominent. This is seen around the portal spaces and the subhepatic and intralobular veins (centrilobular cirrhosis). Its characters are sporadic or irregular distribution and absence of active hyperplasia. It is, therefore, mainly a fibrous replacement and not an active proliferation. As mentioned above, nutmeg livers not infrequently have a naked-eye appearance that suggests cirrhosis, but microscopic examination shews little fibrosis, and that of a sporadic character in no way comparable to cirrhosis. Atrophy of the liver cells and replacement fibrosis are most advanced under the capsule of the liver; macroscopically this subcapsular fibrosis closely resembles chronic perihepatitis. Microscopically, however, the capsule is seen to be unaffected (*vide* Fig. 15).

The morbid anatomy of the other abdominal organs is that seen in the backward pressure of mitral or obstructive lung disease and need not be described in detail. The *spleen* is firm, deeply engorged, but not increased in weight. This is shewn by Kelynack's[1] statistics of the weight of the spleen in 56 cases of nutmeg liver due to cardiac disease and uncomplicated by any infective process, in which the average weight of the spleen was 7·3 ounces, or just about the normal. The *pancreas* may be enlarged from fibrosis and even palpable during life.[2] It may, however, be of normal size.[3]

Clinical Picture.—*Symptoms.*—There is a feeling of tightness, heaviness, and discomfort in the right hypochondrium, which is markedly tender on pressure. It may be noted in passing that the tenderness of an enlarged liver in alcoholic cirrhosis, which is usually explained as being due to active fibrous hyperplasia, may be due to chronic venous engorgement, the result of alcoholic dilatation of the heart. The tenderness may be of three kinds: (i) Superficial, and referred from the distended liver to the 7th to the 10th dorsal skin areas. (ii) Tenderness of the liver itself, which probably depends on the distension and stretching of the capsule of the organ. (iii) Great tenderness accompanied by pain, which is independent of palpation, is due to concomitant perihepatitis and may be accompanied by friction. Mackenzie[4] points out that the tenderness which is usually assumed to be in the liver is often in the abdominal walls, for the area of tenderness is generally more extensive than would correspond to the liver, and the abdominal wall is tender when it is carefully lifted up.

Chronic portal engorgement gives rise to impairment or loss of appetite, dyspepsia, flatulence, tympanites, and favours gastro-intestinal catarrh, constipation, and irregularity of the bowels with perhaps diarrhoea; together they lead to considerable distension of the abdomen. Assimilation is interfered with, and the patients are thin, though this may be somewhat masked by oedema; in children growth is stunted, and there may be very considerable wasting. Haematemesis is usually said to occur, but I have not seen it in uncomplicated cases.

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[1] Kelynack. *Birmingham Med. Rev.*, 1897, xli, 86.

[2] Hale White. *Internat. Clin.*, 1896–97, Series 6, ii, 90.

[3] Lefas. *Arch. gén. de méd.*, 1900, clxxxv, 539.

[4] Mackenzie, J. *The Study of the Pulse*, p. 221, 1902, and *Brit. Med. Journ.*, 1906, i, 1525.

In a case of mitral stenosis[1] with thrombosis of the splenic vein haematemesis occurred, but was evidently due to the extreme venous engorgement of the vasa brevia of the stomach which open into the splenic vein.

Dyspnoea on exertion and in advanced cases orthopnoea, due to the primary cardiac lesion or to concomitant ascites, are common. It is possible that dyspnoea in some instances may be due to hepatic insufficiency, and, like the uraemic form, toxaemic.

Physical Signs.—The skin over the malar bones is much congested and of a high colour. The lips, ears, and skin elsewhere may be cyanosed. The forehead and especially the temples have a slightly icteric tint. The contrast between these two tints of the skin produces a facial aspect which is very characteristic of advanced mitral disease. The slight icteric tinge is probably due to a low grade of catarrhal inflammation of some of the small intrahepatic bile-ducts, but it may be due to oedematous swelling of the mucous membrane of the bile-ducts. Ordinary catarrhal jaundice depending on the spread of gastro-duodenal inflammation to the biliary papilla sometimes supervenes. As a rule the subcutaneous abdominal veins are not dilated, but I have published a drawing of a child shewing considerable enlargement of the veins communicating between the superior and inferior venae cavae.[2]

The *liver* is nearly always enlarged, and can be felt two or three fingers' breadths below the costal arch in the right nipple line. Its size varies with, and is a fair indication of, the condition of the right side of the heart. It diminishes when backward pressure is relieved and is affected in a similar manner by free purgation. The surface of the liver is smooth to the touch and firm. When the engorgement is marked, the liver is tender from distension; the tenderness, like the increased size, is a good index of increased backward pressure. In a small percentage of cases the liver can be felt by the hand to pulsate with each beat of the heart.

In 235 cases of tricuspid regurgitation analysed by Pitt[3] pulsation was present in 15, and in 87 cases of tricuspid stenosis in 8.

With one hand over the liver and the other pressed into the right loin, the liver can be felt to expand and diminish like an accordion. This proves that the pulsation is not a transmitted impulse, but is due to injection of the liver with blood at each beat of the heart. The left lobe pulsates more than the right, as the blood regurgitates into it more readily.

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[1] Green, T. H. *Brit. Med. Journ.*, 1899, ii, 1415.

[2] Rolleston. *Trans. Med. Soc. Lond.*, 1909, xxxii, 19.

[3] Pitt, G. N. *System of Medicine* (Allbutt and Rolleston), 1909, vi, 325.

Pulsation may also be communicated to the liver from a hypertrophied and labouring right ventricle, the liver receiving a jog with each beat of the heart, the left lobe being chiefly affected ; in rare instances pulsation is transmitted from an abdominal or even an hepatic aneurysm.

The following are some of J. Mackenzie's [1] conclusions : Pulsation of the liver occurs only in valvular disease, but is very commonly detected by the polygraph, though not by the hands. When once the liver has begun to pulsate, it probably always continues to do so till death. Pulsation of the liver may be synchronous with the systole of the ventricles or of the auricles, and corresponds with the rhythm of venous pulsation in the neck. Pulsation of the liver synchronous with contraction of the auricle is generally associated with tricuspid stenosis and may be confused with pulsation communicated to the liver from a vigorously contracting heart.

If pressure in an upward direction is made on the liver, the jugular veins in the neck may swell up. The incompetency of the valves at the origin of the jugular veins allows the column of blood in the inferior vena cava, right auricle, and superior vena cava to be displaced upwards.

The enlarged liver may push the diaphragm up and by encroaching on the thorax lead to some collapse of the base of the right lung. Dulness, absence of breath-sounds and of vocal vibrations at the right base behind are not uncommon in advanced mitral disease, and may be due to collapse of the lower lobe of the lung or to a pleural effusion.

Chronic venous engorgement of the liver necessitates chronic portal obstruction ; ascites may thus be set up, and if of long duration is associated with some chronic peritonitis. Whether the latter is set up by the ascites or whether the ascites is the result of the chronic peritonitis brought about by chronic portal and peritoneal engorgement is open to discussion. Probably the general opinion is that chronic peritonitis is the primary and important factor. It must be remembered that in the backward pressure of heart disease not only is the venous pressure in the liver increased, but from obstruction to the passage of lymph out of the thoracic duct into the veins the pressure of lymph in the lymphatics is raised. Further, from stagnation the nutrition of the lymphatics and blood-vessels is apt to become impaired. As a result of these factors ascites might occur in the absence of chronic peritonitis. Ascites occurs in more than half the cases of tricuspid regurgitation, but is less frequent than oedema of the feet.

Thus, in 235 cases of tricuspid reflux collected by Pitt, oedema was noted in 200 and ascites in 140 ; oedema and ascites occurred together in 124 cases, oedema alone in 76, ascites alone in only 14, and neither in 21. This tends to support the view that the ascites of nutmeg liver is part of a general oedema.

This form of ascites resembles that of chronic peritonitis in persisting and recurring, and in not being, like the ascites of cirrhosis, a close forerunner of death. The fluid is usually straw-coloured, but it may be chylous or pseudo-chylous; out of 14 collected cases of milky effusion in heart disease 8 were chylous and 6 were pseudo-chylous (Wallis and Schölberg[1]).

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[1] Mackenzie. *A Study of the Pulse*, 1902, p. 220, Edin. and London.

Dry pleurisy may occur or signs of pleural effusion at the right base; these may be explained as the result of pulmonary apoplexy, which is commoner in the lower lobe of the right lung. On the other hand, the upward projection of the enlarged liver may simulate a small effusion.

The *urine* is diminished in amount, and in advanced cases is concentrated, of a high specific gravity, high coloured, and uratic. It contains urobilin and in some instances haematoporphyrin,[2] but unless there is definite jaundice bile-pigment is absent. In some instances there is albuminuria; its occurrence is variable and does not necessarily depend on gross structural change. When backward pressure falls on the renal veins in a marked degree the nutrition of the renal epithelium may be sufficiently impaired to allow of albuminuria. Alimentary glycosuria is quite exceptional and probably depends on concomitant pancreatic change rather than on hepatic insufficiency. The excretion of urea varies with the amount of urine; diminution in its amount has been correlated with hepatic insufficiency. Leucine and tyrosine have exceptionally been found in the urine, but may be transitory (Dixon Mann[3]). Their appearance can be explained either by autolysis of the liver or by failure of the liver to deal with the amino acids absorbed from the alimentary canal. In an advanced case under my care leucine and tyrosine were absent; the examination was kindly made by Dr. Leathes.

There is delay in absorption of liquid from the intestines, and accordingly the amount of urine excreted during digestion, instead of being larger than that excreted during fasting, the normal relationship, is less (Gilbert and Lereboullet[4]). This has been tested by giving meals with eight-hour intervals and collecting the urine every four hours. The term *opsiuria* has been applied to this condition (Lecerf[5]).

Complications.—Well-marked jaundice may be produced by extension of catarrhal gastritis to the duodenum, and so to the biliary papilla and lower end of the common bile-duct. In rare instances jaundice may be a terminal event due to acute infection falling on the liver. In a case under my care in which jaundice appeared three days before death there were enormous masses of streptococci in the sinusoids of the liver[6]; but it is not necessarily infective, for Oertel[7] describes deep jaundice, due to multiple aseptic necrosis of the cells in the centre of the hepatic lobules, as a terminal incident accompanied by stupor, delirium, and coma (*vide* p. 575).

In chronic venous engorgement of the liver there are several factors

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[1] Wallis and Schölberg. *Quart. Journ. Med.*, Oxford, 1910–11, iv, 170.
[2] Garrod, A. E. *Lancet*, 1900, ii, 1327.
[3] Dixon Mann. *Quart. Journ. Med.*, Oxford, 1907–8, i, 25.
[4] Gilbert et Lereboullet. *Compt. rend. Soc. Biol.*, Paris, 1901, liii, 276.
[5] Lecerf. *Thèse de Paris*, 1901.
[6] Rolleston. *Trans. Med. Soc. Lond.*, 1909, xxxii, 19.
[7] Oertel. *Arch. Int. Med.*; Chicago, 1910–11, vi, 293.

favouring auto-intoxication. (i) In the liver itself. Owing to malnutrition the antitoxic function of the liver cells is impaired or arrested. Toxic bodies derived from the alimentary canal therefore tend to pass into the general circulation and affect the nervous centres. (ii) Owing to portal stagnation digestion is interfered with and more toxic material is carried to the liver, which is, as has already been said, unable to cope with even the normal amount. (iii) Chronic venous engorgement of the renal veins leads to diminished urinary excretion.

Course.—The course of the disease depends on the condition of the heart, and may, like it, improve periodically only to relapse again. The hepatic enlargement tends to increase during each recurrence, though, as already pointed out, the liver gets somewhat atrophied towards the end of a case. According to Mackenzie, pulsation once it appears remains to the end.

Termination.—Death is commonly due to gradually increasing cardiac failure or to some terminal infection, usually of other parts of the body, such as pneumonia or pleurisy. Infection, however, may attack the liver itself, and thus give rise to icterus gravis. In such cases diarrhoea and vomiting are the early symptoms ; jaundice, haemorrhages, and dry tongue develop, and finally delirium, stupor, and coma precede death. A slighter terminal affection may shew itself by acute perihepatitis.

Diagnosis.—In manifest heart disease there will be no difficulty ; but in the class of cases termed by Hanot hepatic asystole, in which attention is focussed on the liver, the condition may appear to be primarily hepatic and be thought to be cirrhosis, or, when the liver is large and very tender, malignant disease.

The abdominal wall rarely shews the dilated veins seen in many cases of hepatic cirrhosis, and there is no enlargement of the spleen apart from embolism, and the symptoms improve on cardiac tonics. In any case of cirrhosis definite signs of mitral disease should lead to revision of the diagnosis; but an apical systolic murmur is not infrequent in cirrhosis, though it is seldom persistent. In cases of hepatic asystole the history, the smooth surface of the liver, the comparatively smaller amount of enlargement, and diminution in size following cardiac tonics, together with absence of severe pain and of cachexia, should enable the observer to eliminate malignant disease. Two cases simulating malignant disease have been under my care (*vide* p. 523).

Treatment may be divided into two heads, which, however, overlap to a certain extent. The first and most important is that of the primary heart disease or of the combined lung and heart affections. Digitalis is the most efficacious drug and may be given as the tincture in x. to xx. minim doses four to six times a day, or the fresh infusion in corresponding doses. Digitalin is not so efficacious as the tincture or infusion. The pill containing digitalis, squill, and mercury (Addison's pill at Guy's Hospital, Baillie's pill at St. George's) is often very successful. Digitalis with citrate of caffeine may also be given. Strophanthus, though well adapted for mitral stenosis, and convallaria are not so powerful for good in tricuspid reflux as digitalis. If there be emphysema and bronchitis, iodide of potassium, carbonate of ammonia, ipecacuanha, may be given in addition. Rest in bed, and if necessary paracentesis of the abdomen or tapping of the legs, are essential parts of the treatment.

In the second place, the engorgement of the liver and its results may be further treated by purgatives, diuretics, and local applications. As purgatives, magnesium sulphate or sodium sulphate should be employed to run off the excess of fluid from the intestinal vessels; mercury in the form of blue pill or calomel is a valuable remedy. Aloes or eau de vie allemande may also be given with advantage. As diuretics the cardiac tonics already mentioned and apocynum may be employed.

The administration of liver substance by the mouth is said[1] to act as a diuretic when the lesion is not too advanced, and to increase the amount of urea and diminish the abnormal constituents, such as urobilin and albumin, present in the urine.

If there be much pain and perihepatitis is present, poultices, hot applications, leeches over the liver, or bleeding may be employed. The diet should be simple, nutritious, and not mainly liquid, as this tends to aggravate the already waterlogged condition of the patient. For sleeplessness morphine is the most satisfactory drug; sulphonal, or preferably trional or veronal, which act more rapidly, chloralamide, or paraldehyde may also be tried, but should not be given constantly; if a sleeping draught is frequently needed, its composition should be changed. Spa treatment at Vichy, Neuenahr, Nauheim, Pougues, etc., may be tried when the patient is sufficiently well to bear the journey.

PERICARDITIC PSEUDO-CIRRHOSIS OF THE LIVER

THIS condition, which for practical purposes may be regarded as an extreme grade of chronic venous engorgement of the liver in cardiac patients with adherent pericardium, was described by Pick[2] in 1896. The clinical features—recurrent ascites, an enlarged firm liver, absent or slight jaundice, and no oedema of the legs—may suggest cirrhosis, inasmuch as during life adherent pericardium may be latent or overlooked. The liver is in an extreme stage of chronic venous engorgement and shews fibrous hyperplasia. Any peritoneal adhesions or thickening that may be present were regarded by Pick as accidental or secondary either to chronic venous engorgement of the peritoneum or to ascites. The patients are chiefly young subjects with a history of rheumatic endocarditis and pericarditis, and without any of the usual antecedents of hepatic cirrhosis.

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[1] Spillmann et Dernang. *Congrès de méd. int.*, Lille, 1899.

[2] Pick. *Ztschr. f. klin. Med.*, 1896, xxix, 385.

This condition merges, on the one hand, into chronic venous engorgement of the liver, with which it is, to my mind, most closely allied; and, on the other, into cases of universal perihepatitis associated with adherent pericardium (*vide* p. 162). Some few adhesions or slight peritoneal thickenings are comparatively frequent in the cases belonging to the group described by Pick; when these peritoneal changes are advanced, the condition ceases to be Pick's pseudo-cirrhosis of pericarditic origin and belongs to that of chronic universal perihepatitis associated with adherent pericardium (multiple serositis). Kelly[1] grouped together under the heading of multiple serositis these cases of Pick's pseudo-cirrhosis and the cases of "iced liver," or, as they are usually called in, England, "chronic universal perihepatitis." To quote his own words:—"Some distinction—anatomically at least—may be drawn between cases in which the lesions are confined to the peritoneum and cases in which lesions are more widespread. The cases in which the pericardium is unaffected reveal no congestive alterations in the liver. Clinically, however, the two classes of cases are very much alike, and the 'Zuckergussleber' may occur in both."

The following case, in which there was some chronic peritonitis, otherwise exactly resembled Pick's pseudo-cirrhosis, but from the presence of peritoneal change it must be regarded as occupying a position midway between pseudo-cirrhosis and chronic peritonitis associated with adherent pericardium (multiple serositis).

A girl aged four was under my care in St. George's Hospital with recurrent ascites, chronic jaundice of slight degree, a large pulsating liver, and pulsating jugular veins more marked on the right side of the neck. The heart was much enlarged, there were systolic and diastolic apical murmurs, and an accentuated pulmonary second sound. At the necropsy the pericardium was universally adherent, the left ventricle was dilated, greatly hypertrophied, and shewed much chronic endocarditis of the musculi papillares. The mitral valve segments were much thickened and the orifice was somewhat dilated. The left auricle was also dilated. The right side of the heart, contrary to what was expected, was not dilated. The heart and pericardium weighed 17 ounces. The hepatic veins were enormously dilated and were each of them as large as the inferior vena cava. The liver was nutmeggy and adherent to the diaphragm. Microscopically the appearances were those in figures 14, 15, namely, sporadic and subcapsular fibrosis and extreme chronic venous engorgement. There were a few adhesions and a few ounces of fluid in each of the pleurae, and general opacity of the peritoneum.

Eisenmenger,[2] who among others disputes the existence of the condition as a distinct morbid entity, believes that in children the vessels of the general systemic circulation have a better tone and are less disposed to allow transudation to occur than in older people, and that the more marked hepatic manifestations are thus accounted for. He further suggests that adhesions in connexion with the adherent pericardium or a concomitant pleural effusion may lead to torsion of the inferior vena cava close to the diaphragm and so set up ascites; and that in other instances local peritonitis in the portal fissure may be the real cause of ascites.

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[1] Kelly, A. O. J. *Am. Journ. Med. Sc.*, 1903, cxxv, 116.
[2] V. Eisenmenger. *Wien. klin. Wchnschr.*, 1900, xiii, 249.

Morbid Anatomy.—The liver is often united to the diaphragm by scattered adhesions. Its general appearance is that of advanced chronic venous engorgement. There is no general chronic peritonitis or universal perihepatitis, but the surface of the liver is usually somewhat irregular, as in ordinary chronic venous engorgement, and often opaque. This opacity is due to fibrosis under the capsule, and on superficial examination resembles chronic perihepatitis, for which it has probably often been taken. On section the liver shews advanced chronic venous engorgement.

The *microscopic appearances* of the liver are those of advanced chronic venous engorgement with very irregularly scattered islands of fibrosis, in which there may be elastic tissue (Carnot and Amet[1]). Much of this apparent fibrosis is due to atrophy of the liver cells, which allows the existing fibrous tissue to come into prominence. There is perhaps a little active fibrous hyperplasia, and, by careful selection, areas resembling multilobular cirrhosis with the addition of chronic venous engorgement can be found. Taken as a whole, however, the amount of fibrosis is scanty, and may be absent in considerable areas. Under the capsule there are extensive atrophy of the liver cells and fibrous replacement. If microscopic examination be limited to a section from this part of the liver, there would appear to be well-marked cirrhosis. But the extent of the fibrous change is confined to a small area under the capsule. It is, however, enough to produce very definite opacity, and, as has already been pointed out, imitates universal chronic perihepatitis. The two conditions are entirely different: in chronic universal perihepatitis (*Zuckergussleber*, "iced liver") the fibrosis is on the outer surface of the capsule. The appearances in subcapsular fibrosis are well shewn in Fig. 15.

Although there is no genuine cirrhosis comparable to portal cirrhosis in cases of ordinary adherent pericardium, well-marked hepatic cirrhosis is found in ~~the majority~~ of cases of calcified adherent pericardium (Diemar,[2] Wells,[3] Mitchell[4]). What relation, if any, exists between calcified pericardium and hepatic cirrhosis is unknown. This condition occurs in adults, while the patients with Pick's pseudo-cirrhosis are usually children. I have examined some of the latter cases, expecting to find an extension of fibrosis from the adherent pericardium along the hepatic veins into the substance of the liver, but have never found any fibrosis, though the inner walls of the hepatic veins and inferior vena cava are opaque and thickened, as is commonly seen in cases of backward pressure. The pericardium is firmly adherent to the heart, and usually to the chest wall as well. These adhesions are usually due to past

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[1] Carnot et Amet. *Arch. de méd. expér. et d'anat. path.*, 1906, xviii, 767.

[2] Diemar. *Ztschr. f. Heilk.*, 1899, xx, 257.

[3] Wells, H. G. *Am. Journ. Med. Sc.*, 1902, cxxiii, 259.

[4] Mitchell. *Trans. Chicago Path. Soc.*, 1911, viii, 109.

Among 89 Collected cases of calcified pericardium
Turner mentions 12 of hepatic Cirrhosis

Turner, H. H. Internat. Clinics 1924 34 ser., IV, 137

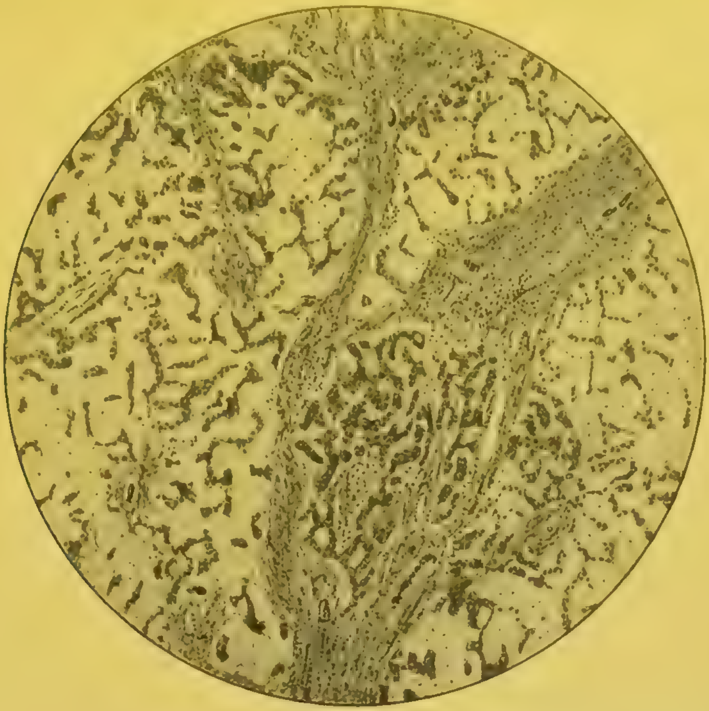

FIG. 14.—Microscopic section from an advanced case of nutmeg liver associated with adherent pericardium (case on p. 97). The liver cells are separated by extravasated blood and compressed and distorted, and there is sporadic fibrosis. This section was specially chosen so as to shew "cardiac fibrosis." × 60.

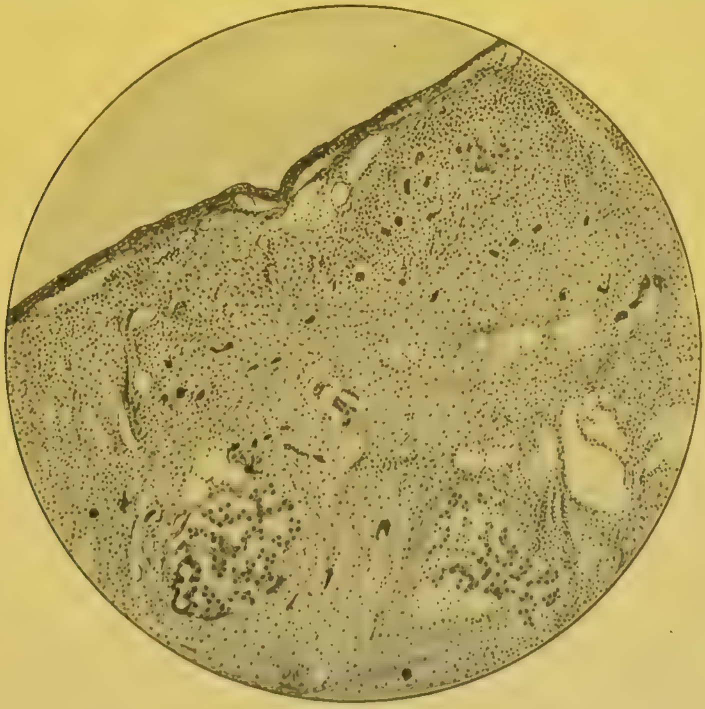

FIG. 15.—Drawing from a microscopic section shewing marked subcapsular fibrosis. The real capsule is seen on the free surface. (From the same case as figure 14.)

rheumatic pericarditis, but in some instances the pericarditis is tuberculous, and under these conditions the liver may also be tuberculous (*vide* p. 101). The tuberculous nature of the adherent pericardium may only be manifest on microscopic examination, as in two cases recorded by Sappington and Rau.[1]

Mechanism of the Morbid Processes.—The pericardial adhesions by contracting lead to dilatation of the right auricle, inferior vena cava, and hepatic veins, and by this means the free regurgitation of blood into the liver is rendered permanent. It is possible that at the time of the primary pericarditis inflammation spreads to the mouths of the hepatic veins, and by weakening their walls leads to dilatation and so to a freer entry of blood into them. When once brought about, this dilatation of the hepatic veins probably becomes permanent. The brunt of backward pressure thus falls on the liver, while the other branches of the inferior vena cava—the renal and iliac veins—suffer less than in ordinary cases of chronic engorgement of cardiac origin.

The **clinical aspect** of the cases has already been referred to, and the absence of oedema of the legs, which may tend to differentiate the condition from ordinary backward pressure of heart disease, mentioned on p. 96. During life the liver may be firm and enlarged, and so may suggest cirrhosis, especially if the presence of valvular disease and an adherent pericardium be overlooked. In most instances the associated cardiac and pericardial disease will be easily detected, and doubt will not arise. It may be necessary to tap the abdomen many times, but the intervals are often prolonged, thus differing from the frequent paracenteses necessary in chronic peritonitis. Jaundice is either absent or slight. The following case illustrates the general features of the condition:

A girl, aged eleven years, was under the care of my colleague, Dr. Penrose, for three years with morbus cordis and ascites, for which tapping had been required about fifteen times. At the necropsy there was mitral and tricuspid incompetence with an adherent pericardium ; the liver—3 pounds 6 ounces—was enlarged and shewed some thickening of its capsule with a few adhesions. The hepatic veins were very prominent but there was no fibrosis around them. The surface of the liver was irregular from depressed lines due to atrophy and fibrous increase around the subcapsular veins. On section the liver was nutmeggy and gave the impression of some fibrous increase. Microscopically there was sporadic fibrosis, but no genuine multilobular cirrhosis, the branches of the hepatic veins and intralobular venules were greatly dilated, and the liver cells atrophied. The appearances were those of advanced chronic venous engorgement with more fibrosis than usual. There was no chronic peritonitis; there were old adhesions in the left pleura and recent ones in the right pleura.

Prognosis and Results.—When the condition of hepatic pseudo-cirrhosis has become established the prognosis is very bad, though life

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[1] Sappington and Rau. *Arch. Pediat.*, N.Y., 1906, xxiii, 814.

Pick advocates the operation of cardiolysis may be prolonged for a considerable time. Tuberculous peritonitis may supervene as a secondary result; this was proved to be the sequence of events in a case recorded by Nachod,[1] in which laparotomy a year before death proved the absence of tuberculosis at that time. Secondary tuberculous infiltration of the portal spaces may occur. The term cardio-tuberculous cirrhosis has been applied to advanced chronic venous engorgement of the liver complicated in this way by tuberculous infection.[2] These cases, which are chiefly met with in children, are associated with tuberculous adherent pericardium and more advanced tuberculous disease elsewhere, especially in the peritoneum and pleura.

These conditions of "hepatic pseudo-cirrhosis" and cardio-tuberculous cirrhosis are closely allied both to nutmeg liver and to the cases of general perihepatitis secondary to adherent pericardium. Clinically the chief difference from nutmeg liver is the absence of any signs of cardiac valvular disease. The proper treatment, however, is that of chronic venous engorgement of the liver, viz. cardiac tonics and diuretics. The treatment suitable for cirrhosis is of no use in these conditions.

INFARCTS

CONTRARY to what might be supposed from the double blood-supply of the liver, from the hepatic artery and portal vein, and their free communication, appearances resembling infarcts in other organs occur in the liver.

There may be (i) true infarcts with necrosis of the liver cells; these are usually anaemic, but in rare instances, as in Versé's and Winternitz's[3] cases, haemorrhagic; or (ii) pseudo-infarcts or sub-infarcts (Adami) in which there is no necrosis of the liver cells. These are much less rare than true infarcts. They are usually haemorrhagic, and are really localised areas of venous engorgement; but in some instances there are anaemic areas without necrosis of the liver cells. To the naked eye true infarcts and pseudo-infarcts resemble each other; the distinction between them is the presence or absence of necrosis of the liver cells. The important factor in causing a true infarct appears to be obstruction or thrombosis of the intralobular branches of the portal vein.

Infarcts and pseudo-infarcts in the liver may be either haemorrhagic or anaemic. The haemorrhagic form is more frequent.

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[1] Nachod. *Prag. med. Wchnschr.*, 1898, xxiii, 330.

[2] *Vide* Moizard et Phulpin, *Arch. de méd. des infants*, 1899; Cousin, *Gaz. hebd. de méd.*, Jan. 14, 1900; Wells, H. G., *Am. Journ. Med. Sc.*, 1902, cxxiii, 299; Grancher, *Journ. de méd. et de chir. prat.*, 1904, lxxiv, 412; Monnier, *Gaz. méd. de Nantes*, 1905, 2. s., xxiii.

[3] Winternitz. *Johns Hopkins Hosp. Bull.*, Balt., 1911, xxii, 396.

In 32 cases collected by Lazarus-Barlow,[1] including Chiari's[2] 17 cases, 28 were haemorrhagic and four anaemic. In 50 cases, including most of Lazarus-Barlow's, 36 were haemorrhagic, 12 were anaemic, and in two cases (Pitt,[3] M'Weeney[4]) haemorrhagic and anaemic infarcts were both present.

Morbid Anatomy.—*Haemorrhagic infarcts and pseudo-infarcts* in the liver are well-defined areas, sometimes square or pyramidal. They are not raised above the surrounding surface ~~of the liver~~; in fact, Zahn spoke of them as atrophic red infarcts. Histologically the liver cells are atrophied, granular, and may contain fat globules, while the nuclei do not stain well. There is, however, no coagulation-necrosis. The capillaries are engorged with blood. There may be considerable leucocytic infiltration of the affected area.

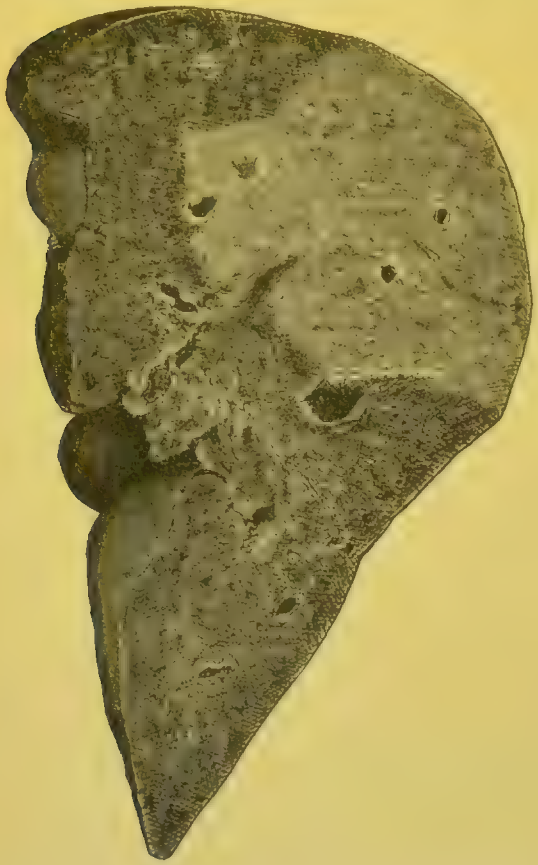

FIG. 16.—Anaemic infarct in the liver from a case with thrombosis of the intrahepatic branches of the portal vein and an empyema. St. George's Hospital Museum, Series ix, 165c. (Drawn by L. Jones, M.S.)

In *anaemic infarcts and pseudo-infarcts* the affected area is of a whitish-yellow colour and is sharply marked off from the surrounding healthy liver substance. There may be, but is not necessarily, a zone of congestion immediately at its junction with the healthy liver tissue. The area has much the same appearance, only it is larger and more prominent, as the white patches seen on the surface of the liver in many infectious diseases, which on microscopic examination shew local fatty change. At the margin of the infarct there are leucocytes, red blood-corpuscles, and altered blood-pigment.

As a result of infarction a slight amount of localised replacement fibrosis may result, but probably not enough to give rise to a cicatrix. Frerichs[5] described depressed areas of atrophy on the surface of the

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[1] Lazarus-Barlow, W. S. *Brit. Med. Journ.*, 1899, ii, 1342.

[2] Chiari. *Ztschr. f. Heilk.*, 1898, xix, 475.

[3] Pitt, G. N. *Trans. Path. Soc.*, Lond., 1895, xlvi, 75.

[4] M'Weeney. *Journ. Path. and Bacteriol.*, Cambridge, 1912, xvi, 403.

[5] Frerichs. *Diseases of the Liver*, ii, 396. Transl. New Sydenham Soc., 1861.

often only found on section of the organ and do not
appear on the surface, and when seen externally are

liver, producing lobulation, as due to thrombosis of branches of the portal vein; it is conceivable that in their earliest stages these areas were infarcts. Infarct of the liver cannot be recognised clinically and is only of pathological interest.

Etiology.—The conditions which have been thought to play a part in producing true and false infarction of the liver are: (1) Obstruction of the branches of the portal vein, either by embolism or thrombosis; (2) obstruction of the branches of the portal or hepatic veins by new-growth; (3) obstruction of both the portal and hepatic veins at the same time; (4) embolism of the hepatic artery; (5) endarteritis of the hepatic artery; (6) retrograde embolism of the hepatic veins; (7) severe trauma.

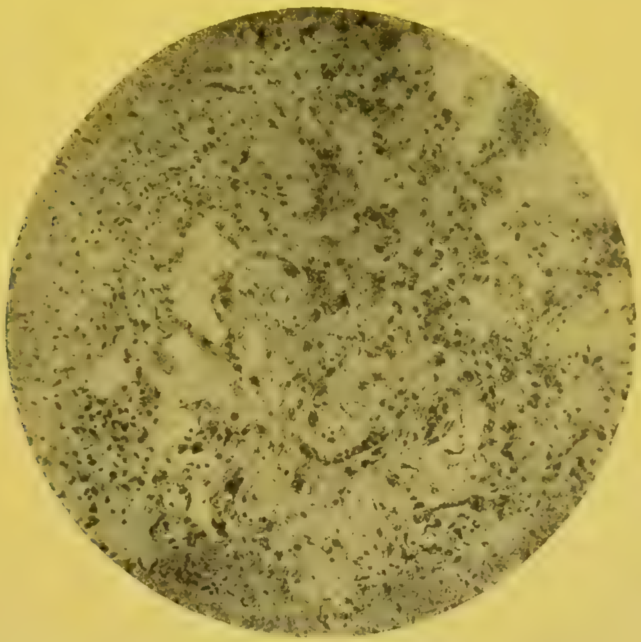

FIG. 17.—Photomicrograph of a true infarct in the liver. In part of the section the liver cells have lost their outline and their nuclei only remain. (Dr. S. G. Penny.)

(1) *Embolism and Thrombosis of the Branches of the Portal Vein.*—The most frequent vascular lesion found in cases of infarcts in the liver is ante-mortem blood-clot in the intrahepatic branches of the portal vein, either embolic and conveyed from thrombosis in its tributaries, or formed *in situ.*

Embolism.—Chiari records 15 cases of haemorrhagic areas in the liver due to embolism. This number is comparatively large and rather contrasts with the isolated observations of others, which, though less numerous, point to thrombosis of the vein as a more frequent antecedent. Chiari's cases, however, include embolism with fragments of new-growth as well as with detached blood-clot.

Thrombosis.—Thrombosis of branches of the portal vein, without any other vascular obstruction, such as embolism of the hepatic artery or thrombosis of the hepatic veins, has been found in cases of hepatic infarction by Osler,[1] Pitt,[2] Chiari,[3] and others. I have seen this association on several occasions.

A man, aged forty-six, died on May 3, 1902, in St. George's Hospital with a right-sided empyema and a dilated heart; there had been no abdominal symptoms and no ascites. The main trunk of the portal vein had an adherent parietal thrombus, and some of the branches of the portal vein in the right lobe of the liver were occluded with firm clots. There were two anaemic infarcts, one as large as one's hand (*vide* Fig. 16), the other a quarter of that size. The veins going to these areas were thrombosed. The hepatic artery was free from embolism, thrombosis, and arteriosclerosis. The infarcted areas were distinctly raised above the level of the surrounding liver substance. Although the macroscopic appearances were those of an anaemic infarct, the microscopic were those of a haemorrhagic infarct, the capillaries being full of blood. It is possible that in this case backward pressure from the dilated heart and infection played some part, in addition to portal thrombosis, in inducing the infarctions.

A man, aged forty-eight, under the care of Sir W. Bennett and myself, was operated upon for a localised subphrenic abscess due to a perforated gastric ulcer. Thrombosis of the right popliteal vein appeared before death, and at the necropsy thrombosis of a branch of the right division of the portal vein with a sharply marked haemorrhagic area was found in the corresponding part of the liver.

(2) *Obstruction of Branches of the Portal or Hepatic Veins in the Substance of the Liver by New-growth.*—Both these conditions may be associated with infarction of the liver. When the intrahepatic branches of the portal vein are obstructed by growth the process is usually embolic and due to small emboli of infective cells derived from a carcinoma in the stomach, colon, or other organ within the territory of its tributaries. When the hepatic veins are occluded, the growth may invade them from without, spread through the capillaries from embolic growths in the portal vein, or in rare cases be due to retrograde embolism (*vide* p. 53).

In a woman, aged forty, who died in St. George's Hospital with a spindle-celled sarcoma surrounding the pancreas, there were secondary growths in the liver which blocked up some branches of the hepatic veins and gave rise to haemorrhagic infarcts in the corresponding areas. There were anaemic infarcts in the spleen due to blocking of the splenic vein. There was no evidence of arterial embolism. Longcope[4] describes an anaemic infarct associated with thrombosis of the hepatic vein, and a haemorrhagic infarct associated with thrombosis of the portal vein; in both Longcope's cases the thromboses were secondary to gastric carcinoma.

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[1] Osler. *Trans. Assoc. Am. Phys.*, 1887, ii, 136.

[2] Pitt. *Trans. Path. Soc.*, 1895, xlvi, 75.

[3] Chiari. *Ztschr. f. Heilk.*, 1898, xix, 475.

[4] Longcope, W. T. *Univ. Penn. Med. Bull.*, 1901, xiv, 223.

(3) *Combined Portal and Hepatic Vein Thrombosis.*—Thrombosis of the portal vein may, when combined with thrombosis of the hepatic veins, give rise to infarction of the liver. Thus, Pitt[1] records a case of very widespread thrombosis in the abdominal aorta, splenic, left renal, and right middle cerebral arteries, in the right hepatic and the branches of the portal veins. In Pitt's and in M'Weeney's cases, in which the portal and hepatic veins were thrombosed, there were both anaemic and haemorrhagic infarcts in the liver. But in a case of combined hepatic and portal vein thrombosis that I examined there was no appearance of infarction in the liver.[2]

(4) *Embolism of the hepatic artery* is very rare. It has been found both in man and experimentally to lead to necrosis of the liver (*vide* p. 47). Embolism of its branches may give rise to anaemic infarcts.

In a case of embolism of the bifurcation of the hepatic artery by a vegetation derived from the aortic valves C. Ogle[3] found anaemic infarcts in the liver. In a case of aortic and mitral disease Baldwin[4] found between 20 and 30 anaemic infarcts in the liver which were genuinely necrotic. The corresponding branches of the hepatic artery contained old thrombi. There were infarcts in the spleen, kidneys, brain ; so there is little doubt that the infarcts in the liver were embolic.

(5) *Endarteritis obliterans* of the branches of the hepatic artery has been described by Bonome[5] as the cause of haemorrhagic and of necrotic infarcts in the liver in cirrhosis. In an extensive anaemic infarct in a case of eclampsia O'Sullivan found hyaline degeneration of the walls of the capillaries of the hepatic artery.

(6) *Retrograde Embolism.*—Embolism of one of the hepatic veins by a clot derived from the heart which had worked its way against the flow of blood—or retrograde embolism—has been observed to be associated with an hepatic infarct (Arnold[6]).

(7) *Trauma* may lead to the production of appearances resembling infarcts in the liver.

Traumatic Haemorrhagic Infarcts.—In rare instances trauma may lead to the passage of part of the liver through a hole or rupture in the diaphragm. If the piece of liver becomes strangulated, haemorrhage takes place into its substance and the appearance resembles that of a haemorrhagic infarct.

In a case recorded by C. Ogle,[7] part of the left lobe of the liver, measuring 5 by 4 inches, was herniated through the diaphragm, and to the naked eye resembled the section of a "cardiac" spleen. Microscopically the liver tissue was disorganised and contained much blood pigment, red blood-corpuscles, and the remains of liver cells, but differed considerably from the appearances in a haemorrhagic pseudo-infarct of the organ.

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[1] Pitt. *Trans. Path. Soc.*, 1895, xlvi, 75.
[2] Rolleston. *Ibid.*, 1899, l, 148.
[3] Ogle, C. *Ibid.*, 1895, xlvi, 73.
[4] Baldwin, F. A. *Journ. Med. Res.*, 1904, viii, 431.
[5] Bonome. *Arch. di biol.*, Firenze, 1899, liii, 319.
[6] Arnold. *Virchows Arch.*, 1891, cxxiv, 388.
[7] Ogle, C. *Trans. Path. Soc.*, Lond., 1897, xlviii, 114.

Traumatic Anaemic Infarcts.—Severe trauma may cut off the vascular supply to a part or parts of the liver. If life is prolonged for some days, the areas of the liver thus deprived of their blood-supply present to the naked eye an appearance resembling an anaemic infarct. The areas are firm and white in colour and have been regarded as infarcts by Klebs,[1] Lubarsch,[2] Lazarus-Barlow, Gruber,[3] Heile.[4] Microscopic examination shews that there is undoubted necrosis in these areas and that they correspond to true infarcts in the kidney and spleen.

(8) There may be no manifest vascular obstruction. In such cases a septicaemic or toxaemic condition is possibly responsible for the lesion. I have seen an infarct in the liver without any evidence of portal thrombosis or embolism of the hepatic artery. The patient, a man aged twenty-five, died with a localised peritonitis in the lesser sac due to acute pancreatitis.

Pathogeny.—Cohnheim and Litten[5] injected coarsely powdered chromate of lead and wax into the portal vein and produced portal thrombosis but no infarcts. Zahn,[6] however, found that sterilised mercury injected into the mesenteric veins was followed after an interval of eight days by haemorrhagic infarcts. But in actual practice thrombosis of the portal vein or its branches is so frequently seen without hepatic infarction that some other factor would appear to be necessary. Rattone suggested that the additional factor is blocking of the hepatic artery, but this is hardly ever found to be the case; Chiari and Steinhaus[7] argued in favour of a feeble circulation and low blood-pressure in the hepatic artery associated with venous congestion. Kohler[8] thought that backward pressure in the hepatic veins was a necessary adjunct, but this, again, is not borne out by the facts of morbid anatomy. It is probable, therefore, that infarction of the liver is not solely a mechanical process.

By the injection of tissue fibrinogen Wooldridge[9] produced portal thrombosis with haemorrhages and necrotic areas in the liver. Wooldridge's results suggest that, in addition to vascular disturbances, such as thrombosis or embolism of the blood-vessels of the liver, a toxaemic or septicaemic condition is necessary or at least very favourable to the production of infarction in the liver. Possibly a low arterial blood-pressure, which is favoured by septicaemia, may, when combined with an infective or toxic blood state, be sufficient to produce hepatic infarction.

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[1] Klebs. Virchows *Festschrift*, 1891, S. 8.

[2] Lubarsch. *Fortschr. d. Med.*, Berl., 1893, xi, 805.

[3] Gruber. *Frankfurter Ztschr. f. Path.*, 1912, x, 442.

[4] Heile. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1900, xxviii, 443.

[5] Cohnheim und Litten. *Virchows Arch.*, 1876, lxvii, 153.

[6] Zahn. *Centralbl. f. allg. Path.*, 1897, viii, 860.

[7] Steinhaus. *Deutsch. Arch. f. klin. Med.*, 1904, lxxx, 364.

[8] Kohler. Quoted by Welch, Allbutt's *System*, 1899, vi, 280.

[9] Wooldridge, L. C. *Trans. Path. Soc.*, Lond., 1888, xxxix, 421.

The importance of an infective condition is supported by the fact that Apert[1] produced multiple anaemic infarcts, resembling the early stage of abscess formation, in the liver, heart, and kidneys of a rabbit by injecting pus containing colon bacilli and numerous anaerobic micro-organisms into the vein of the ear.

ACUTE CONGESTION OF THE LIVER

Active acute congestion of the liver is, of course, the first stage of acute hepatitis, which may, like similar processes elsewhere, abort and be followed by a rapid return to the normal condition. It is therefore impossible to draw a hard and fast line between the clinical manifestations of active congestion and of acute hepatitis in an early stage. Acute congestion of the liver is both more frequent and of more importance in warm countries, where malaria and dysentery are rife, than in temperate countries.

From a systematic standpoint the **causes** of active congestion of the liver may be first divided into:

(I) Active vaso-dilatation of the capillaries of the hepatic artery; this may be reflex and due to the effects of cold applied to the surface of the body. This is the mechanism by which chills produce active congestion of the liver. In England cold does not act in this way to anything like the same extent that it does in tropical climates, where the resistance of the liver is often much diminished from past attacks of congestion or inflammation in connexion with malaria and dysentery. In persons who have had dysentery or repeated attacks of malaria exposure to cold readily produces active congestion of the liver; this is sometimes spoken of as "a chill on the liver." But all cases described in these words are not thus explained; the effects of excessive eating and drinking, dyspepsia, gastro-intestinal catarrh, and constipation are sometimes described in these euphemistic terms. Exposure to high temperature in the tropics is, especially if followed by an accidental chill, a cause of acute congestion of the liver. Direct nervous stimulation has been suggested to explain active congestion of the liver in diabetes, but the voracious appetite of diabetic patients probably accounts for any active hepatic congestion. Experimentally, however, irritation of the "diabetic centre," as by Bernard's puncture experiment, gives rise to intense hyperaemia of the liver.

(II) The more reasonable and by far the commonest cause of active hepatic congestion is to be found in the toxic bodies reaching the organ through the blood-stream. This may occur (*a*) through the hepatic artery, as in fevers, especially malaria, and various infective conditions, though in the latter the process may pass on into inflammation or suppuration. It probably occurs in the early stages of icterus gravis, and no doubt is partly responsible for the enlargement of the liver seen in that disease and in the early stage of acute yellow atrophy.

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[1] Apert. *Bull. Soc. Anat.*, Paris, 1900, lxxv, 204.

Experimentally it has been shewn that subcutaneous or intravenous injection of the extract obtained from muscular tissue gave rise to intense congestion of the liver in animals (Richet [1]).

(*b*) But generally the poison is derived from the gastro-intestinal tract and reaches the liver by the portal vein. It therefore follows overeating, and especially alcoholic excess, gout, gastritis, indigestion, dysentery. Physiologically active congestion of the liver occurs in digestion and is heightened by spices, pepper, mustard, and curries; in great excess such condiments may no doubt in a suitable subject set up acute and active hepatic congestion. Alcohol is an important factor, especially in hot climates, in producing acute congestion; this probably explains why hepatitis is seventeen times commoner in Europeans than among the natives of India (Davidson [2]). Other toxins may be carried by the portal vein, as in constipation, or, to take a more extreme case, in phosphorus poisoning. The suppression of menstruation (Senator [3]) or of habitual haemorrhages from piles, and the climacteric, have been thought to give rise to active hyperaemia of the liver, but the evidence is somewhat slender.

Morbid Anatomy.—The liver is enlarged, dark in colour, the uniform congestion contrasting with the partial engorgement seen in the chronic venous stagnation of mitral disease. The liver cells shew cloudy swelling, and may be fatty and pigmented, and the capillaries are dilated and full of red blood-corpuscles. The small bile-ducts may shew proliferation of their lining epithelium (cholangitis), which accounts for jaundice when present.

Clinical Picture.—The symptoms vary. Sometimes the patient is so ill that he must remain in bed; in other cases exercise can quite well be taken. There is malaise, with headache, giddiness, mental depression, irritability, insomnia, and gastro-intestinal symptoms, such as loss of appetite, a foul tongue, a bad taste in the mouth, sometimes nausea, flatulence, and constipation. There is a feeling of discomfort and weight in the hepatic region, and pain in the right shoulder. The conjunctivae become muddy and slightly icteric, the face is often congested, and the skin shares in these changes and may be irritable. The bowels are irregular; there may be diarrhoea or constipation. Epistaxis sometimes occurs.

The symptoms of acute congestion of the liver are much the same as some of those described by Murchison as lithaemia and regarded by him

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[1] Richet, C. *Acad. des sciences*, Paris, Dec. 31, 1900; *Progrès méd.*, 1901, p. 23.

[2] Davidson. Article "Tropical Liver," in *System of Medicine* (Allbutt and Rolleston), 1907, ii, part 2, 574.

[3] Senator. *Berlin. klin. Wchnschr.*, 1872, ix, 615.

As a good substitute for Carlsbad salts a dram of a mixture of sodium sulphate 2 parts, sodium bicarbonate 1 part and sodium chloride (1 part) should be dissolved in a pint of water and sipped on an empty stomach in a hot [illegible] as possible, a third of the whole being sipped in 3 periods with 20 minutes interval. x

x Vide Manson's Tropical Diseases p. 494, 1921 as due to functional inadequacy of the liver. The liver is usually enlarged and may project two or three fingers' breadths below the costal arch in the nipple line. It is decidedly tender on palpation. As a result of repeated attacks of hepatic congestion piles may be produced. Some writers describe oedema of the feet in acute hepatic congestion.

The urine is high coloured, uratic, concentrated, and of high specific gravity. The amount of urobilin is increased. The amount of ammonia in the urine is little, if at all, above the normal, and thus contrasts with the increase of ammonia found in the urine of patients with cirrhosis (Bain [1]). If there is jaundice, bile-pigment will be found in the urine. Albuminuria only occasionally occurs; it is transitory and can be explained as the result of poisons passing from the bowel and failing to be stopped by the liver; the kidneys are thus affected, and toxic albuminuria results.

The term *tropical liver* describes the more or less chronic condition found in persons who have had many attacks of acute congestion in hot climates. According to Cantlie, there is usually a history of malaria, diarrhoea, dysentery, or long-standing indigestion. Anaemia, weakness, nervous irritability, and irregularity of the bowels are essential features. The spleen is generally enlarged in addition to the liver. Probably some of these cases are in the early or pre-ascitic stage of cirrhosis.

Duration.—Acute congestion when treated usually lasts about a week, but may, if neglected, become chronic, and may then be a preliminary stage to cirrhosis.

Treatment.—In marked cases the patient should be confined to bed and kept on a milk diet. If, as sometimes happens, ordinary milk cannot be borne, skimmed milk, junket, milk jelly, or whey should be tried, and, if necessary, chicken broth or the juice of raw meat should be given. Plenty of water should be allowed, but alcohol must be strictly prohibited. Intestinal antiseptics, such as fractional doses of calomel, perchloride of mercury, guaiacol, or salol, should be given and the bowels kept freely open by calomel and salines, such as Carlsbad or Epsom salts and mineral waters. By free purgation the congestion is greatly relieved, as shewn by diminution in the size and disappearance of tenderness of the liver. Chloride of ammonium in xx.-grain doses has been credited with the power of reducing the congestion and preventing inflammation and suppuration. At the outset, if there are symptoms of gastritis, a soothing mixture containing bismuth and bicarbonate of sodium should be given.

Local Applications, etc.—For the pain in the hepatic region a large linseed poultice may be applied, and if it gives relief, be changed every three hours. Turpentine stupes, cold compresses, or dry cupping may be employed if poultices fail. If there is pain on respiration, resembling that of pleurisy and due to perihepatitis, the side should be strapped as for fractured ribs. Leeches applied over the hepatic region or at the margin of the anus have been employed to deplete the congested liver. Pain is undoubtedly relieved by leeching the skin over the liver, although it is hardly probable that much blood is withdrawn from the liver through dilated venous anastomoses between the veins of the abdominal wall and the parumbilical vein of the falciform ligament. Neither is it likely that leeches applied to the anus draw off much blood from the portal vein. Venesection from the veins of the elbow was formerly much in vogue in hepatic congestion, but has almost completely gone out for many years. Direct aspiration of the liver and abstraction of blood is highly spoken of by many. In performing this risky operation the puncture of the liver may have to be performed several times before a vessel of sufficient size to bleed freely is struck. This withdrawal of blood from the liver is by no means a harmless proceeding. If a large branch of the portal vein or the inferior vena cava is wounded, fatal haemorrhage may take place into the peritoneal cavity. Indian surgeons, such as Hatch[1] and Maitland,[2] who have had fatal experience of aspirating the liver, condemn the procedure. Cantlie,[3] by limiting the length of the aspirating needle to 3½ inches or so, considers that the danger of wounding the inferior vena cava is obviated.

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[1] Bain. *Brit. Med. Journ.*, 1898, ii, 941.

When the more acute symptoms have passed off, the patient's diet should be improved, and tonics, such as strychnine in an acid mixture, should be given. When convalescent, the patient will be benefited by change of air. A cool, somewhat high, inland resort is usually most suitable. The seaside, especially when the patient lives near the shore, is often harmful and gives rise to constipation and so to a return of hepatic congestion.

In the slighter forms of active congestion of the liver due to over-eating, drinking, and aggravated by constipation and a sedentary life, free purgation with blue pill and haustus sennae, followed by a simple diet and exercise, should be enjoined. Exercise on horseback or on a bicycle will in many cases act like a charm.

BRIDES/A

Treatment at a spa, such as Vichy, Ems, Neuenahr, Carlsbad, Marienbad, Harrogate, ~~Leamington~~, Llandrindod Wells, has a beneficial effect both by reducing congestion of the liver through purgation and by preventing indiscretions in diet, while the freedom from business worries has a good influence on the mental condition.

Prophylaxis.—In gouty patients care as to diet should be taken to prevent undue hepatic congestion; but it is chiefly in persons who are, or have lived, in the tropics and have suffered from malaria that active congestion of the liver is likely to occur, and in whom, therefore, special care should be taken to avoid causes, such as chills and alcoholic or dietetic excess, which bring on this condition.

Exposure to east wind, sudden changes of temperature, cold draughts, and the risk of chills should be avoided by a malarial patient. The parts of the body exposure of which to cold is most liable to give rise to

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[1] Hatch, W. K. *Ind. Med. Gaz.*, 1898, xxxiii, 137.

[2] Maitland. *Brit. Med. Journ.*, 1902, i, 458.

[3] Cantlie, J. *Ibid.*, 1903, ii, 656.

to Oideum roseum
non-liquefaciens (ZENONI)

ZENONI. Sperimentale FIRENZE, 1912, LXV
33

hepatic congestion are the abdomen, the back of the neck, and the legs and feet (Brunton). These parts should be suitably protected; a good plan is to wear a knitted cholera belt over the abdomen, which should always be changed after free action of the skin. Alcohol is best avoided, and if taken at all should be in small quantities well diluted at meal times. Made dishes, much meat, pastry, and sauces should be avoided.

Constipation should be guarded against by diet, viz. fruit and vegetables, porridge, and a sufficiency of water. If necessary, small doses of calomel or blue pill followed by a saline should be given. Active exercise, not merely walking, but riding, climbing, and other forms of exertion, such as rowing, skipping, which lead to compression of the liver between the diaphragm and the abdominal wall, a kind of natural massage, should be taken by persons whose strength and physique are suitable.

ACUTE HEPATITIS

THERE are a large number of different conditions in which the liver is acutely inflamed. At the outset a division into suppurative and non-suppurative hepatitis may conveniently be made. The suppurative forms of hepatitis are described elsewhere under other headings, such as abscess, pylephlebitis, cholangitis. Acute non-suppurative hepatitis may chiefly attack the liver cells, as in icterus gravis and in acute yellow atrophy, or the brunt of the change may fall on the connective-tissue framework, as in the hepatitis seen after scarlet fever (Klein[1]). (*Vide* also p. 191.) In malaria the liver cells are affected, but not in the same degree as in icterus gravis. The focal necroses seen in typhoid fever, puerperal eclampsia, and in some other infections and intoxications are allied to acute parenchymatous hepatitis, and represent a patchy or localised distribution of the process, which when diffuse gives rise to acute yellow atrophy. Hepatitis may also be due to influenza.

Acute active congestion of the liver and non-suppurative hepatitis are often two stages of the same process, and any attempt to distinguish sharply between them clinically is difficult or impossible.

Morbid Anatomy.—So many different forms of pathological change are included under the title of "acute hepatitis" that no one description can possibly be inclusive. The appearance of the liver varies according to the nature and intensity of the inflammation. In the more acute changes affecting the cells of the liver the condition will resemble that seen in icterus gravis or in acute yellow atrophy, while in less severe types of inflammation the aspect of the liver will resemble that in active congestion (*vide* p. 108). In acute hepatitis as seen in pyaemia the lobules of the liver may be very sharply marked out as bile-stained areas surrounded by greyish-white rings.

[1] Klein. *Trans. Path. Soc.*, 1877, xxviii, 439.

In a patient who had had a prolonged attack of dysentery the liver was deeply congested, oedematous, and almost fluctuating when it was seen during an operation undertaken by Bérard [1] under the idea that there was an abscess. The liver was explored in four places; blood alone came out and the patient rapidly recovered.

The liver of severe malarial infection, which is a definite form of acute inflammation, is usually swollen, enlarged, and more or less pigmented, from a brownish to a slaty black tint. The outline of the lobules is indistinct. In more chronic malarial infection two forms of hepatitis, described by Kelsch and Kiener,[2] should be mentioned here: (i) Hyperémie phlegmasique, in which the enlarged and soft liver shews perihepatitis, cloudy swelling, and karyokinesis of the liver cells with small-celled infiltration of the portal spaces; (ii) nodular parenchymatous hepatitis, in which there is hyperplasia of the liver cells forming small nodules on the surface of the organ, which may resemble multiple adenomas or even masses of secondary new-growth, though they are not umbilicated.

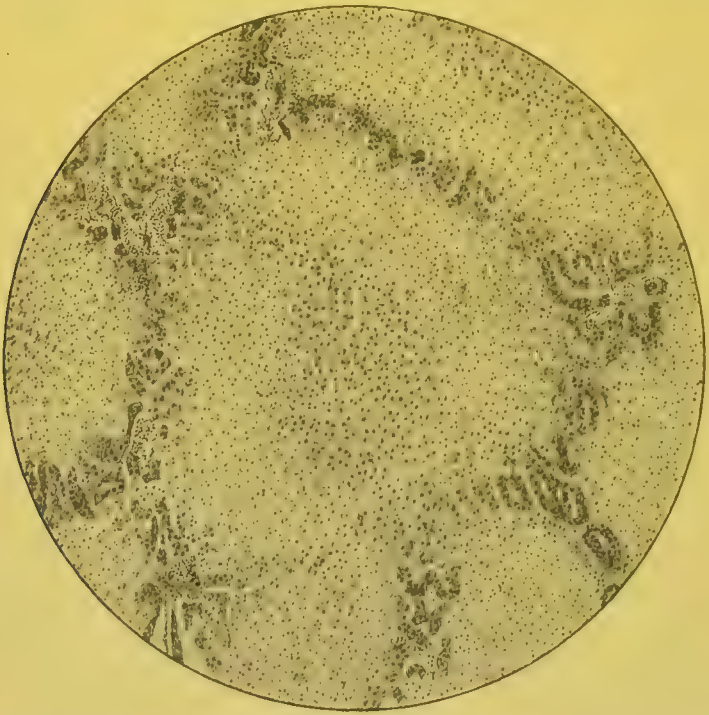

FIG. 18.—Drawing from a microscopic section from a case of pyaemia due to acute necrosis of both fibulae. Shews cloudy swelling and necrosis of the liver cells in the intermediate zone of the liver, and small-celled infiltration at the margin of the lobule.

Attention may also be drawn to a rare form of *acute parenchymatous hepatitis* which shews in a marked degree compensatory hyperplasia of the liver cells. The condition is very closely allied to the nodular

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[1] Bérard. *Lyon méd.*, 1902, xcviii, 752.

[2] Kelsch et Kiener. *Arch. de physiol. norm. et path.*, 1878, 571; 1879, 354.

parenchymatous hepatitis just described, but is not confined to malarial infection, and resembles the condition of the liver in subacute atrophy (*vide* p. 583).

I described a case of this kind in 1892,[1] but the Morbid Growths Committee of the Pathological Society regarded the condition as allied to cirrhosis. Prof. S. Delépine communicated to me full details of a case of acute interstitial and parenchymatous inflammation with imperfect regeneration of the liver cells, forming tubules containing plugs of bile, and giving rise to hyperplastic nodules. The patient, aged eleven years, was under Sir Lauder Brunton's care in 1887 for jaundice after one of the acute specific fevers; from this recovery occurred, but six months later the jaundice recurred and proved fatal. The liver, 44 oz., shewed yellow areas of nodular hyperplasia in which the cells had undergone fatty degeneration and so led to the fatal result.

In this condition of acute parenchymatous hyperplasia of the liver cells with symptoms of subacute atrophy there may either be complete jaundice, no bile passing into the intestine (Widal and Abrami[2]), or the stools may be coloured. The larger bile-ducts are healthy, and jaundice has been thought to be due to pressure on the minute bile channels exerted by the enlarged liver cells (Ramond and Géraudel[3]).

The *histological appearances* in acute hepatitis also necessarily vary in the different forms. They may be those already described in acute congestion of the liver, viz. dilatation of the capillaries, acute lymphangitis, cloudy swelling, fatty degeneration and pigmentation of the liver cells, and catarrhal inflammation of the small bile-ducts. In addition the liver cells may shew focal necroses, with thrombosis of small vessels, and in some instances a varying degree of small-celled infiltration. The small cells are partly polymorphonuclear and other leucocytes, and partly young connective-tissue cells, and are found in the areas of focal necrosis and at the periphery of the lobules.

In some cases there is a diffuse small-celled infiltration, which is, like the hepatitis of congenital syphilis, unicellular. Figure 19 from a case of acute hepatitis accompanied by fever and jaundice and without any suppuration of the liver illustrates this condition. Cases of *subacute diffuse hepatitis* are sometimes seen, and may be regarded in the light of a very acute form of cirrhosis or midway between portal cirrhosis and acute yellow atrophy (Symmers[4]).

In the liver of severe malaria there are, in addition, many large phagocytes containing the parasites and pigment. As well as leucocytes, the endothelial cells of the vessels and Kupffer's star-like cells act as phagocytes.[5] The liver cells undergo degeneration and contain pigment, granules of bile and of haemosiderin. There is little fatty change in the cells. Dilatation of the capillaries may lead to the production of areas resembling cavernous tissue (Ewing[1]).

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[1] Rolleston. *Trans. Path. Soc.*, Lond., 1892, xliii, 81.
[2] Widal et Abrami. *Bull. et mém. Soc. méd. des hôp. de Paris*, 1905, 3. s., xxv, 523.
[3] Ramond et Géraudel. *Ibid.*, 1905, 3. s., xxv, 600.
[4] Symmers, D. *Am. Journ. Med. Sc.*, Phila., 1908, cxxxv, 251.
[5] See Barker. *Johns Hopkins Hosp. Rep.*, Balt., 1895, v, 221.

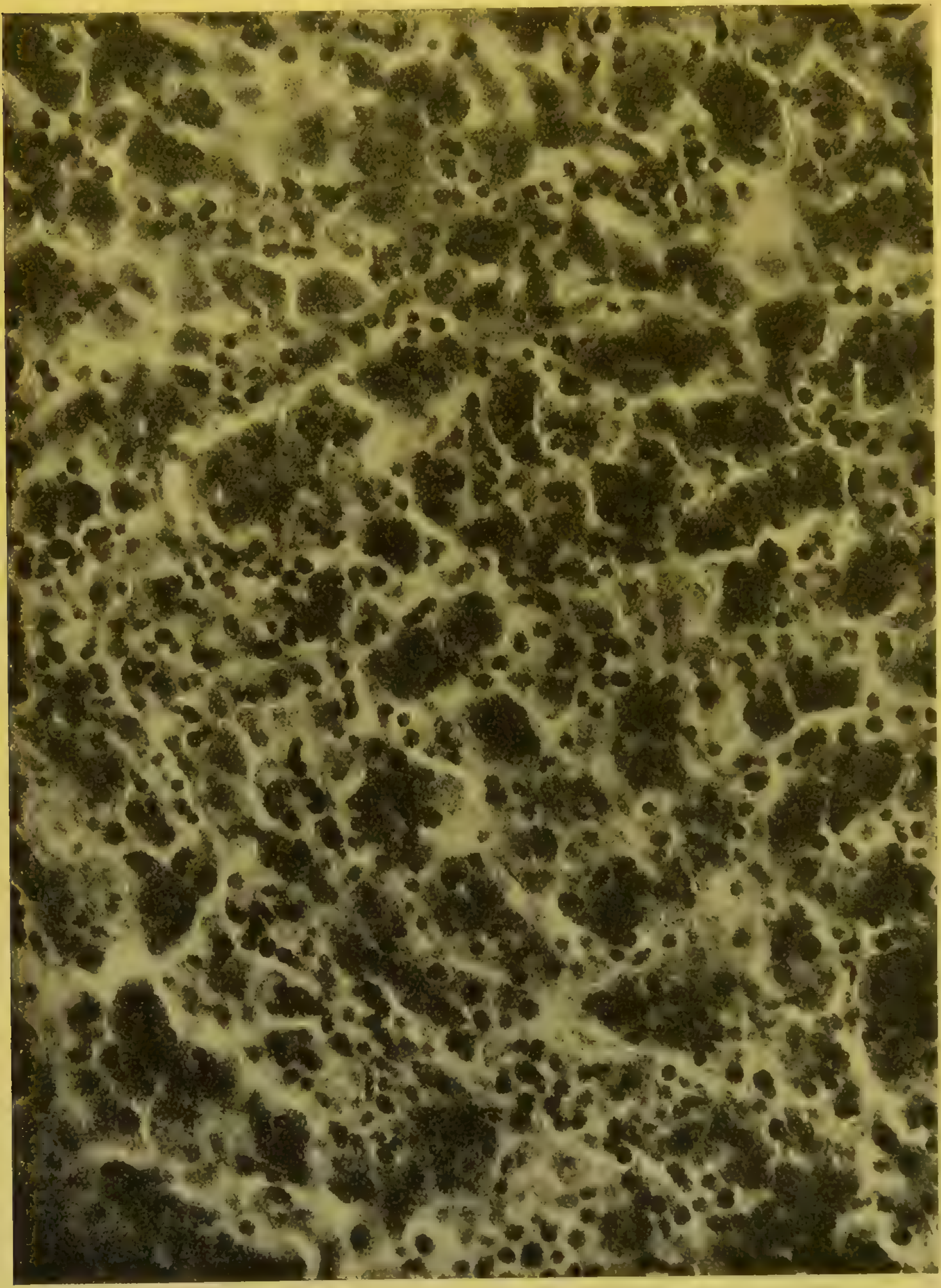

FIG. 19.—Photomicrograph of acute hepatitis. The liver cells are separated from each other by small round-celled infiltration. From a section prepared by Dr. Bolam. (Photomicrograph by Dr. H. Spitta.)

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[1] Ewing. *Journ. Exper. Med.*, N.Y., 1901–5, vi, 154.

Clinical Picture.—Acute non-suppurative hepatitis due to a general infection or toxaemia may shew itself by jaundice. If the constitutional symptoms accompanying the jaundice are severe, the condition resembles that described under icterus gravis. With a milder grade of clinical manifestations, the jaundice is spoken of as being infectious or toxaemic, or may conform to the type of Weil's disease. When hepatitis due to a general infection is not accompanied by jaundice, there may be little to attract attention to the liver, though on examination it may be found to be somewhat enlarged and tender. The condition, which in this country is, perhaps rather vaguely, usually spoken of as acute hepatitis, occurs in patients who have suffered from frequent attacks of malaria, from dysentery, or from both diseases. As the result of exposure to cold or of excess in eating or drinking they suffer from the train of symptoms described later on.

Clinically acute hepatitis is seldom seen in England except in persons who have returned from the tropics. It is an acute form of the reaction to irritants which in temperate climates is chronic and eventually gives rise to cirrhosis. The irritant causing hepatitis is in many instances derived from the gastro-intestinal tract. The symptoms are the same as those of active congestion of the liver, to which reference should be made, but are more marked. The temperature in hepatitis is raised, and on the degree of fever a clinical distinction has been drawn between active congestion and hepatitis; Cantlie [1] considers that a temperature above 100° shews that the condition is inflammatory, and one below this point, active congestion. Davidson,[2] however, fixes the dividing line at 102° F. The gastro-intestinal symptoms described in acute congestion are accentuated and vomiting is more frequent. The bowels may be constipated, or there may be diarrhoea; in both cases the motions are very offensive. The pain in the shoulder is more marked than in simple congestion, and from extension of hepatitis to the capsule of the liver the case may become complicated by perihepatitis. This makes the descent of the diaphragm painful and leads to shallow respirations. The diminished movement of the diaphragm can be seen by x-rays. The patient is irritable, low-spirited, and sits up in bed. The abdomen is rigid and the liver is uniformly enlarged and tender. When there is concomitant malaria, the spleen is also enlarged. Toxic albuminuria, which has been spoken of as hepatogenic (Teissier [3]), is occasionally present.

Diagnosis.—When there is distinct jaundice, a diagnosis must be made in the most severe cases from acute yellow atrophy and from phosphorus poisoning. In acute yellow atrophy the rapid course, the presence of nervous symptoms, and the fact that the liver, if at first increased in size, soon diminishes, are characteristic. In phosphorus poisoning the history and the haemorrhagic condition of the vomit and stools are points of importance. In acute atrophy and phosphorus poisoning the constitutional symptoms are far more severe than in acute hepatitis. But the distinction between acute hepatitis with jaundice and subacute atrophy of the liver may be very difficult. When the jaundice is slight, infectious jaundice should be thought of.

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[1] Cantlie. *Encyclopaedia Medica*, Edin., 1901, vii, 10.

[2] Davidson. Article "Tropical Liver," *System of Medicine* (Allbutt and Rolleston), 1907, ii, part ii, 576.

[3] Teissier, J. *Les Albuminuries curables*, p. 34, 1900, Paris.

In some cases in which the liver is very considerably enlarged and the other symptoms severe it may be very difficult or impossible to be sure that there is not suppuration in the liver, especially as acute hepatitis may be the first stage of a tropical abscess. This difficulty not uncommonly occurs in malarial patients. According to Rogers,[1] leucocytosis occurs in amoebic hepatitis but differs from that in ordinary suppuration in not shewing any relative increase in the polymorphonuclears ; it then resembles amoebic abscess (*vide* p. 137). In 5 out of 15 cases of amoebic hepatitis recorded by Rogers, exploration for abscess gave negative results.

Bérard's case, already referred to, is an example of the difficulty in making a diagnosis between acute hepatitis and hepatic abscess. Remlinger,[2] Bozzolo,[3] and others describe a form of acute hepatitis under the name of "the infective liver" which clinically imitates hepatic abscess.

The same difficulty may arise in differentiating the condition from more diffuse forms of hepatic suppuration, such as suppurative pylephlebitis and cholangitis. In these cases some time may be required before an accurate differential diagnosis can be made.

The cases of subacute diffuse hepatitis described by Symmers merge clinically into the conditions of acute cirrhosis and of subacute atrophy of the liver.

The **treatment** is on the same general lines as in simple congestion, but is rather more active. The patient should be kept in bed on a low diet, and the local pain and tenderness over the liver relieved by cold applications, poultices, scarifying the skin, leeching, dry cupping, or by strapping the hepatic region with narrow strips of plaster as if for fractured ribs. Milk, or the modifications of it mentioned on page 297, is the staple diet, but if it disagrees, raw meat-juice, chicken broth, jelly, or other similar preparations should be substituted. Plenty of water should be given or its equivalent, such as soda water or lemonade. Alcoholic drinks of all kinds should be strictly tabooed.

The bowels should be kept freely open by salines, such as sulphate of sodium or of magnesium, or calomel may be given with advantage. Vomiting and signs of gastritis should be allayed by bismuth, hydrocyanic acid, bicarbonate of sodium, etc. Diarrhoea, unless excessive, should not be checked. Large doses of ipecacuanha, 20 to 40 grs. once or twice a day after a dose of tincture of opium, are invaluable in amoebic hepatitis,

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[1] Rogers, L. *Med.-Chir. Trans.*, Lond., 1907, xc, 163.

[2] Remlinger. *Presse méd.*, 1903, i, 86.

[3] Bozzolo. *Riv. crit. di clin. med.*, 1902, iii, 169.

The hypodermic injection of $\frac{1}{2}$ gr of emetine hydrochloride, twice daily for 6 days, has been ~~found by Rogers to act~~ is most efficacious~~ly~~ in curing amoebic hepatitis (Rogers, Hill

This form of treatment has taken the place of large doses of Ipecacuanha powder (20-40 grs) and ammonium chloride

Rogers. Brit. med. journ., 1912, i, 1424

Hill Lancet, London 1916, i, 504

~~whether there be dysentery or not~~, as a means of preventing suppuration (Rogers[1]). Chloride of ammonium in twenty-grain doses should be given three times a day during the acute stage, and may be combined with dilute hydrochloric acid and a few minims of liquor strychninae. If there is a history or evidence of malaria, quinine should be given as soon as the gastro-intestinal symptoms have subsided.

The abstraction of 10 to 15 ounces of blood from the substance of the liver by means of an aspirator is much advocated and has been thought to prevent the further development of suppuration. It is, however, not entirely without risk, as fatal haemorrhage into the peritoneal cavity has been known to follow[2] (*vide* p. 110). When convalescent, the patient should go to a cool, fairly bracing climate, and will get benefit from treatment at a spa, such as Harrogate, ~~Carlsbad, Marienbad, Neuenahr, Ems~~, Vichy. The prophylactic treatment is that of acute congestion (*vide* p. 110).

HEPATIC ABSCESS

INTRAHEPATIC suppuration may be conveniently divided into two main groups: (I) The large, single, or "tropical" abscess, and (II) multiple abscesses. Strictly speaking, this classification is not entirely satisfactory; for occasionally there may be two large abscesses in the liver, and a single abscess may infect the remainder of the liver and give rise to several secondary abscesses; while, on the other hand, multiple abscesses may unite and eventually form a large areolar abscess. It has, however, a practical advantage, for cases of large single or tropical abscess are accompanied by more characteristic signs and are in general curable ~~by operation~~, whereas multiple abscesses are less easy to diagnose and cannot be benefited by surgical treatment.

SINGLE OR TROPICAL ABSCESS

Etiology.—*Dysentery.*—The causation of tropical abscess is more closely related to dysentery than to any other condition. In some statistics there is a close association between the diseases termed dysentery and the subsequent development of hepatic abscess. Azevedo Sodré,[3] adding together the statistics of Moore, Macpherson, Marshall, Morehead, and Dutrouleau, found that in 1997 necropsies on dysentery there were 407 hepatic abscesses, or 20 per cent. In 314 selected cases of hepatic abscess taken by Kelsch and Kiener,[1] 268, or 75 per cent, had had dysentery. In a later analysis by Kelsch[2] of 500 cases of hepatic abscess dysentery was present in 85 per cent. On the other hand, E. J. Waring's[3] statistics from India do not shew such a close relation; thus, in 2758 cases of dysentery treated in the Madras Presidency between the years 1826 and 1843 there were 68 cases of abscess, or 2·5 per cent; whereas in his 300 fatal cases of abscess, only 82, or 27 per cent, were admitted for dysentery. Buchanan's[4] figures also shew a want of parallelism between the curves of the incidence of dysentery and of hepatic abscess in India. Thus, the natives suffer more severely from dysentery and less from hepatic abscess, while the converse holds good among European soldiers.

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[1] ~~Rogers, *Med.-Chir. Trans.*, Lond., 1907, xc, 145.~~

[2] Hatch, *Ind. Med. Gaz.*, 1898, xxxiii, 137; Maitland, *Brit. Med. Journ.*, 1902, i. 458.

[3] Azevedo Sodré. *XXth Century Practice of Medicine*, 1899, xvi, 253.

In 79,723 cases of dysentery among the natives there were 127 cases of hepatic abscess, or 1 in 628; while in the European army during four years (1893–96) there were 7972 cases of dysentery and 441 cases of abscess, or 1 in 18.

The following arguments have also been urged against the too exclusive view of the dysenteric origin of hepatic abscess:—

(i) That hepatic abscess is much commoner among English soldiers in India than in other tropical countries, such as Jamaica, where dysentery is as frequent as it is in India.

(ii) That an increase in the curve of dysentery—for example, in war-time—is not accompanied or followed by any increase in the liver abscess curve.[5]

In the bacillary dysentery of Japan Shiga has never seen hepatic abscess as a complication, and in the dysentery seen in the South African War, 1899–1902, which was not amoebic, hepatic abscess was very rare. In 250 cases observed by Washbourn and Richards[6] at the Imperial Yeomanry Hospital, Deelfontein, there was only one hepatic abscess; out of 466 cases of dysentery treated at the Imperial Yeomanry Hospital, Pretoria, there were two fatal cases of pylephlebitis, and another case, in which after an operation for piles performed some six months after an attack of dysentery, a single hepatic abscess developed and was successfully operated upon. While in South Africa (1901) I made enquiry from medical officers, who all agreed that single hepatic abscess as a sequel of South African dysentery was extremely rare. In 1906 a man who was in South Africa in 1901, but had not been in any other foreign country,

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[1] Kelsch et Kiener. *Traité des maladies des pays chauds*, Paris, 1889.

[2] Kelsch. *Sem. méd.*, 1900, xx, 80.

[3] E. J. Waring. *Abscess in the Liver in the East Indies*, p. 123, 1854.

[4] Buchanan, W. J. *Journ. Trop. Med.*, 1899, i, 173.

[5] Editorial, *Ind. Med. Gaz.*, 1902, xxxvii, 193.

[6] Washbourn and Richards. *Brit. Med. Journ.*, 1900, ii, 668.

or by the patient ^
having been a
carrier only (Low).
Rogers has shewn that
in single hepatic abscess
the dysentery is very often
latent, whereas in severe
sloughing dysentery the
multiple amoebic abscesses
in the liver do not give
rise to any clinical
features.

Low. *Brit. Med. Journ.*, 1916, ii, 867.

DURAND. *Ibid.*, 1913, CXXI, 583

MALLORY. Journ. Amer. Med. Assoc.,
1920, LXXV, 1714

came under my care with a single hepatic abscess. In 422 cases of dysentery in Rhodesia there were 11 cases of hepatic abscess (Curtis[1]).

Dysentery in temperate climates occurs sporadically as "ulcerative colitis" and in epidemics in asylums. Vedder and Duval[2] have shewn that asylum or institution dysentery is due to a bacillus identical with that described by Shiga and Flexner as the cause of acute epidemic dysentery. Liver abscess is very rare after asylum dysentery. In Gemmel's[3] 80 cases examined after death at the Lancaster Asylum there were two cases; in the epidemics of dysentery at Millbank, 1840–47, W. Baly[4] did not find any case of abscess.

(iii) That in some cases of hepatic abscess there is no history of dysentery, and that after death the colon may appear quite healthy.

In 456 cases of fatal hepatic abscess recorded in the annual reports of the sanitary commissioner of India during the five years 1896 to 1900 the intestines were free from ulceration in 47 per cent. Charles[5] quotes similar statistics.

In the cases of amoebic abscess in which the colon is normal at the necropsy[6] it is possible that the amoebae gain entrance to the portal system by some minute lesion which subsequently heals over.

The absence of any history of dysentery in some cases of hepatic abscess may be explained by its having remained latent. In cases in which evidence of dysentery is found at the necropsy, and in which no history has been forthcoming, the original attack may have been forgotten, since an abscess may occur years, even ten;[7] after dysentery, or may be ~~remembered and~~ described as diarrhoea.

twenty (Low),
twenty nine (Durand)
or 30 (MALLORY)

(iv) It has been thought that the dysenteric ulceration of the colon is secondary to the abscess. The older observers believed that pus passed down the bile-ducts into the intestine and set up diarrhoea and ulceration. This explanation is no longer accepted. But that absorption from the abscess may set up diarrhoea and intestinal ulceration is shewn by some cases of fatal hepatic abscess in which a recurrence of dysentery or recent ulceration is seen in a colon with evidences of old dysentery. Flexner,[8] indeed, considers that even in amoebic dysentery the intestinal lesions may be secondary to the amoebic hepatic abscess.

(v) Another view is that the two diseases are both independent results of the same infection. In favour of this it has been urged that when dysentery breaks out, patients may develop hepatic abscess who have not shewn any signs of dysentery, while those who contract dysentery do not necessarily become the subjects of hepatic abscess.

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[1] Curtis, H. *Clin. Journ.*, Lond., 1907, xxxi, 57.
[2] Vedder and Duval. *Journ. Exper. Med.*, 1901–5, vi, 181.
[3] Gemmel. *Idiopathic Ulcerative Colitis*, p. 29, 1898, Lond.
[4] Baly, W. *Lond. Med. Gaz.*, 1847, xxxix, 441.
[5] Charles. *Brit. Med. Journ.*, 1908, ii, 1235.
[6] Buxton, J. T., *Proc. Phila. Path. Soc.*, 1899, ii, 49; Flexner, S., *Am. Journ. Med. Sc.*, 1897, cxiii, 553; Thompson, *Manchester Med. Students' Gaz.*, 1903, p. 146; Futcher, *Journ. Am. Med. Assoc.*, 1903, xli, 480.
[7] Josserand. *Lyon méd.*, 1897, lxxxvi, 421.
[8] Flexner. *Amer. Journ. Med. Sc.*, 1897, cxiii, 553.

The, at first sight, puzzling difference in various parts of the world between the relation of the disease called dysentery and the subsequent development of a single hepatic abscess, may be explained in the following way: Under the heading dysentery at least two distinct forms of colitis have been included in the past: (*a*) amoebic dysentery of a chronic type, which is followed by single or tropical hepatic abscess, and (*b*) acute bacillary dysentery, which is not followed by single hepatic abscess. Bacillary dysentery was described in Japan by Shiga,[1] where it was extremely severe, by Flexner[2] in Manila, by Castellani[3] in Ceylon, by Duval and Vedder[4] in America, and by Kruse[5] in Germany. Amoebic and bacillary dysentery may occur side by side; and a patient with acute bacillary dysentery may contract amoebic dysentery. The two diseases were studied and could be distinguished from each other in Manila during the Spanish-American War. This is well shewn by Craig's[6] contrast of 60 cases of amoebic dysentery with hepatic abscess in 22, or 36·6 per cent, and 60 cases of bacillary dysentery, with multiple abscesses in 2 cases.

Tropical dysentery is more often followed by abscess than the dysentery seen in temperate countries, because it is more often amoebic. In 2590 cases of tropical dysentery hepatic abscess occurred in 507 (Councilman and Lafleur[7]). Few statistics are available to determine the incidence of hepatic abscess in amoebic dysentery. Davidson[8] says one-twelfth of those suffering from amoebic dysentery develop abscess. In 119 cases of amoebic dysentery treated in the Johns Hopkins Hospital 27, or 22·6 per cent, had hepatic abscess (Futcher[9]); in Strong's 79 necropsies on cases of amoebic dysentery 14 had liver abscess.

Other Causes of Single or Tropical Abscess.—Apart from dysentery the factors which can be regarded as having a causal relationship to hepatic abscess are comparatively insignificant.

Trauma may give rise to hepatic abscess either (i) directly—for example, when a penetrating wound introduces micro-organisms into the liver substance—or (ii) indirectly, by so reducing the resistance of the liver that any pus-producing micro-organisms which happen to reach the liver either from the general circulation by means of the hepatic artery, or from the alimentary canal, are then enabled to grow and multiply. Thus a blow on the liver may produce a small rupture inside the liver without damaging the capsule; recovery should then occur, but if micro-organisms have previously gained access to the damaged part of the liver, an abscess may follow.

(i) Penetrating wounds with a dagger, knife, or bullet may introduce

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[1] Shiga. *Centralbl. f. Bakteriol. u. Parasit.*, 1898, xxiii, 549; xxiv, 817, 870, 913.

[2] Flexner. *Johns Hopkins Hosp. Bull.*, 1900, xi, 37; *Brit. Med. Journ.*, 1900, ii, 20; *Univ. Penn. Med. Bull.*, 1901.

[3] Castellani. *Journ. Hyg.*, Camb., 1904, iv, 495.

[4] Duval and Vedder. *Journ. Exper. Med.*, 1901–5, vi, 181.

[5] Kruse. *Deutsche med. Wchnschr.*, 1900, xxvi, 637.

[6] Craig. *Amer. Journ. Med. Sc.*, Phila., 1904, cxxviii, 145.

[7] Councilman and Lafleur. *Johns Hopkins Hosp. Rep.*, 1891, ii, 67.

[8] Davidson. *System of Medicine* (Allbutt and Rolleston), 1907, ii, part ii, 598.

[9] Futcher. *Journ. Am. Med. Assoc.*, 1903, xli, 480.

Exploratory puncture ∧
may convey pus from
an empyema into
the liver

In children trauma has been ∧
thought to have a special
etiological importance; Legrand
out of 112 cases of intrahepatic
suppuration in children found
19 due to injury

Legrand. Arch. de méd. des enfants, Par., 1906, IX, 120 micro-organisms, or infection may occur later. A bullet may carry some of the patient's clothing, and thus micro-organisms, into the wound. Infection may also supervene either from the wound or from the blood-stream.

It appears probable that a foreign body may remain encysted in the liver for a considerable time and that suppuration may eventually supervene around it. In such cases it is probable that this part of the liver, having its resistance diminished, becomes infected more easily by the blood-stream. It is hardly likely that micro-organisms introduced at the same time as the foreign body—a bullet, for example—have remained latent. In rare instances, a pin, needle, or fish-bone ~~may~~ penetrate the alimentary canal ~~and~~ enter the liver ~~substance~~, thus giv~~ing~~e rise to ~~hepatic~~ abscess.

(ii) Trauma without any penetrating wound may be followed by hepatic abscess. This is not common. F. C. Turner[1] in 1882 was only able to refer to 12 cases, but this estimate ~~must~~ not ~~be taken as~~ actually represent~~ing~~ its incidence. On the other hand, it may be merely that after trauma a latent abscess increases in size or by leakage sets up a perihepatic or subphrenic abscess.

Extension of Inflammation from Adjacent Parts.—This method of formation of a single hepatic abscess is very rare, and not of much importance. Calculi in the gall-bladder may, by giving rise to ulcerative cholecystitis and perforation, set up suppuration in the adherent liver (*vide* p. 619).

Perforation of a simple or of a malignant gastric ulcer into the liver may set up suppuration in a comparatively limited area. In these cases the abscess cavity may shew necrotic changes which, according to Gilbert and Lippmann,[2] depend chiefly on anaerobic micro-organisms.

I have seen 4 cases in which malignant disease of the cardiac end of the stomach grew directly into the left lobe of the liver and gave rise to somewhat diffuse suppuration in its substance. In one of these cases death was caused by peritonitis due to leaking from the abscess. In a man aged forty-nine years, who died in St. George's Hospital in 1906, and whose chief symptom was grave anaemia, there was an abscess the size of a tangerine orange in the left lobe of the liver; this communicated by a large opening, with thickened edges, with the pylorus and the first part of the duodenum. Microscopically the growth was a columnar-celled carcinoma. The abscess cavity contained blood-clot.

An empyema or an abscess in the base of the right lung, suppuration in or around the right kidney, or a localised subphrenic abscess have also been described as extending into and giving rise to suppuration inside the liver. But in such cases it is not always certain whether the suppuration began in the organ named and extended into the liver, or whether it originated in the liver.

Enteric fever in very rare instances is followed by a solitary hepatic abscess. It is said to be less rare in the tropics than in temperate countries (Bertrand and Fontan [1]), and possibly this is due to the influence of dysentery or to the diminished resistance of the liver induced by dysentery, malaria, and alcoholism. A solitary abscess following enteric fever may be due to infection from an abscess elsewhere, such as suppuration of the parotid, and is rare in uncomplicated enteric fever.

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[1] Turner, F. C. *Trans. Path. Soc.*, 1882, xxxiii, 177.

[2] Gilbert et Lippmann. *Bull. et mém. Soc. méd. des hôp. de Paris*, 1903, 3. s., xx, 871.

In 2000 fatal cases of enteric fever examined at Munich Hölscher [2] found 12 cases of solitary hepatic abscess. W. W. Keen [3] collected 16 additional cases, some of which were secondary to abscesses elsewhere ; Louis's case—one of the earliest on record—and Osler's [4] had parotitis, and Chvostek's perichondritis of the larynx. In 1911 von Eberts [5] collected 30 cases in association with enteric fever ; in 9 cases the typhoid bacillus was isolated from the abscess, in the remaining 21 it was not proved to have a causal relation.

Typhoid bacilli can give rise to a solitary hepatic abscess, but, as a rule, there is a mixed infection.

In two cases of solitary abscess following typhoid fever bacteriological examination gave a pure culture of typhoid bacilli in one ; in the other typhoid bacilli and staphylococci were present (Cassuto [6]).

I have seen one case of a single hepatic abscess after enteric fever.

An Imperial Yeomanry trooper, aged thirty years, who had never had dysentery or been previously out of England, was inoculated against enteric fever in 1900 when on his way to South Africa. He had two attacks of enteric fever in the next twelve months in Africa ; when convalescent from the second attack he had acute pneumonia on the right side, followed by signs suggesting empyema. The chest was aspirated and 12 ounces of sanious pus containing liver cells, but perfectly sterile, were evacuated. Mr. D. Drew excised part of the sixth rib and freely opened an abscess in the anterior part of the right lobe of the liver. The patient was invalided home, and was seen in December 1901 in perfect health.

Influenza.—Tédenat [7] recorded 4 cases of single hepatic abscess arising about twenty-five to thirty days after influenza ; there was no other cause forthcoming. The pus was sterile. Influenza may light up a latent abscess.

Suppurating Hydatid Cyst, etc.—When a hydatid cyst suppurates the conditions are for all practical purposes the same as a single hepatic abscess. In rare instances a round worm in the intrahepatic bile-ducts may give rise to a single small abscess, but, as a rule, there is suppurative cholangitis with multiple suppurating foci.

Infective Granulomas.—Suppuration commonly occurs in actinomycosis

[1] Bertrand et Fontan. *L'Hépatite suppurée*, Paris, 1895.
[2] Hölscher. *München. med. Wchnschr.*, 1891, xxxviii, 43.
[3] Keen, W. W. *Surgical Complications and Sequels of Typhoid Fever*, p. 246, 1898.
[4] Osler. *Studies in Typhoid Fever, Johns Hopkins Hospital*, p. 380, 1895.
[5] Von Eberts. *Amer. Journ. Med. Sc.*, Phila., 1911, cxli, 803.
[6] Cassuto. *Thèse de Paris*, 1900.
[7] Tédenat. *Gaz. hebd. d. méd.*, Paris, 1902, vii, 1073.

Preble states that B. typhosus has been isolated in 12 cases only, in 8 in pure culture and in 4 in association with various staphylococci and streptococci.

ble, R B.
Trans Assoc Am. Phys., Phila., 1913, XXVIII, 572

Bruggeman in 1917 | ∧
collected 23 cases |

O. Bruggeman. Ann. Surg.,
1917, LXV, 462

(*vide* p. 383), and in rare instances supervenes in gummas (*vide* p. 356) and in tuberculomas (*vide* p. 345).

Single Pyaemic Abscess.—A single hepatic abscess is sometimes due to causes, such as general haemic infection, or infection of the portal system, which, as a rule, give rise to multiple abscesses. In exceptional instances suppuration or infection elsewhere in the body is the only cause to account for a single hepatic abscess. Thus, it has followed cutaneous abscesses, whitlow, suppuration of the parotid, bronchiectasis (Muir[1]), parametritis and pyosalpinx (Roughton[2]), prostatic abscess (Lancereaux[3]), scarlet fever.[4]

A single hepatic abscess may also occur as the result of emboli derived from the portal vein or its tributaries. Thus, in exceptional instances it may follow gastric ulcer (Dalton[5]), appendicitis, ulcers in the colon, or ligature of piles.

A single loculated or areolar abscess may be the late stage of a number of neighbouring areas of multiple diffuse suppuration, due to pylephlebitis or suppurative cholangitis affecting a comparatively large intrahepatic branch of the portal vein or bile-duct. What appears to be a single hepatic abscess may therefore occur as the result of portal infections which usually produce multiple hepatic abscesses.

In appendicitis, a single hepatic abscess is very rare indeed; Elsberg[6] in 1906 collected 15 undoubted cases, and in 1911 Quénu and Mathieu[7] accepted 14 only. It sometimes happens that a single abscess is diagnosed and operated upon, but the patient dies, and in the absence of a necropsy the actual condition remains doubtful; when a necropsy is made, multiple abscesses are usually found.

A man had an appendicular abscess, and the appendix was removed; thirteen days later an amoebic abscess of the liver, which was probably present at the time of the first operation, was opened, and after an interval of twenty-eight days more pus was evacuated through the wound of the second operation. Curtis[8] inclined to the view that the appendicitis was subsequent to the hepatic suppuration, and compared the sequence of events to that of appendicitis secondary to gall-bladder infection (Dieulafoy.[9] *Vide* also p. 604).

In the following case a large abscess due to the union of originally separate abscesses was due to infection from the appendix:

A girl aged seven years who, except for a stitch in the right side of the abdomen for more than a year, had never had any signs of appendicitis, came under observation with signs of right-sided pleurisy. Subsequently hepatic abscess was diagnosed, and several operations with the evacuation of pus from the liver were performed. At the necropsy the vermiform appendix was surrounded by old adhesions and recent lymph, and was perforated by a pin. The portal vein was healthy. The liver, greatly enlarged, contained a loculated area of suppuration as large as one's fist in the upper and back part of the right lobe. In the immediate neighbourhood there were spreading foci of suppuration. The rest of the liver was of a bright yellow colour resembling that seen in phosphorus poisoning.[1]

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[1] Muir. *Edin. Hosp. Rep.*, 1894, ii, 100.

[2] Roughton. *St. Barth. Hosp. Rep.*, 1885, xxi, 173.

[3] Lancereaux. *Traité des maladies du foie et du pancréas*, 1899.

[4] Guy's Hospital Museum, No. 1294.

[5] Dalton, N. *King's Coll. Hosp. Rep.*, 1896, ii, 25.

[6] Elsberg. *Ann. Surg.*, 1906, xliv, 220.

[7] Quénu et Mathieu. *Rev. de chir.*, Paris, 1911, xxxi, 519.

[8] Curtis. *Trans. Med. Soc. Lond.*, 1906, xxix, 353.

[9] Dieulafoy. *Presse méd.*, Paris, 1903, p. 448.

Disposing Factors.—*Geographical Distribution.*—Large hepatic abscess is a disease of tropical climates, and cases seen in this country are usually in persons who have been in hot climates and suffered from dysentery, or even from a previous hepatic abscess abroad. Although a tropical disease, hepatic abscess is not met with uniformly in different parts of the tropics; thus, while it is frequent in India, Senegal, Mauritius, Algiers, Egypt, Java, Sumatra, Mexico, Peru, Chile, it is much less common in the West Indies, China, Brazil, Guiana, Ceylon.

Altitude.—In parts of the tropics where the elevation is sufficiently high to reduce the mean temperature to that of a temperate climate, hepatic abscess does not occur.

Climate.—A high mean temperature very decidedly favours the production of hepatic abscess; but an important factor is the occurrence of sudden variations. From the incidence of chills, the commencement of the cold season has been said to be a favourite time for the development of hepatic abscess. But from an analysis of the 236 cases of hepatic abscess in Calcutta Rogers[2] found that there was no special seasonal prevalence.

Acclimatisation.—It would appear that persons who have recently become resident in tropical climates are more likely to suffer from hepatic abscess than those who have been there some time.

In 114 cases E. J. Waring[3] found that 22 per cent of cases of abscess occurred in India in soldiers who had been less than one year in the country, 10·5 per cent in the second year, and 10·5 per cent in the third year. Figures quoted by Davidson,[4] however, shew that, although the mortality is very high in the first year, long residence increases the liability of Europeans in India to abscess.

Race.—Europeans are much more liable to hepatic abscess in the tropics than the natives.

In 79,723 cases of dysentery among the natives of India collected by W. J. Buchanan[5] there were only 127 cases of hepatic abscess, or 1 in 628 cases, whereas in 7972 cases of dysentery among Europeans in India there were 441 cases of hepatic abscess, or 1 in 18.

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[1] Rolleston. *Trans. Path. Soc.*, Lond., 1898, xlix, 106.
[2] Rogers, L. *Brit. Med. Journ.*, 1902, ii, 844.
[3] Waring, E. J. *Hepatic Abscess*, p. 113, 1854.
[4] Davidson. *System of Medicine* (Allbutt and Rolleston), 1907, ii, part ii, 591.
[5] Buchanan, W. J. *Journ. Trop. Med.*, 1899, i, 173.

But when natives give way to alcohol they are more likely to be attacked by abscess. It is said to be commoner in the rich than in the poor natives of India.

The greater susceptibility of the European male to hepatic abscess does not depend, at any rate entirely, on the fact that he is not acclimatised to tropical climates, for hepatic abscess is very rare in European women and children in the tropics, though they are equally subject to dysentery ; further, it does not depend on a special liability to dysentery, for in India the natives are more subject to dysentery and less to hepatic abscess than Europeans. The factors which account for the greater liability of European males to hepatic abscess will be referred to again on p. 126.

Occurrence in England.—As already mentioned, most of the cases observed in England have come from tropical climates, but in a certain number of instances the patient has never left this country. An amoebic liver abscess has been recorded in a patient who had never been out of England (Saundby and Miller[1]). The cases seen in ordinary hospital practice are few, probably not more than two or three a year for each of the large London hospitals.

In sixteen years there were 13 fatal cases of single hepatic abscess at St. George's Hospital ; in 3 of these the fatal abscess was a recurrence, the patients having been previously successfully operated upon. Ten of the cases, all men, had been abroad, 8 in India, 1 in China, and 1 in South Africa. In 15 fatal cases at Guy's Hospital in twenty years, 5 had been in China, India, or in the West Coast of Africa, but in 10 there was no such history, and several patients had never been out of England (Hilton Fagge[2]).

Alcoholism, by depressing the resistance of the liver, disposes to hepatic abscess. Abscess is rare in total abstainers, and, conversely, natives who drink may suffer from liver abscess while their fellows who are teetotallers escape.

According to Sandwith,[3] hepatic abscess is met with only in those natives of Egypt who are addicted to alcohol. In 40 cases tabulated by E. J. Waring[4] only 13, or 32·5 per cent, were sober and temperate ; the remainder, 67·5 per cent, were intemperate. In 170 cases investigated by Megaw[5] 119, or 70 per cent, habitually took alcohol, whilst 51, or 30 per cent, did not.

A stimulating and excessive *diet*, by giving rise to considerable physiological congestion, which under the influence of the tropical climate and a sedentary life may become pathological, prepares the way for hepatitis and so for abscess. As a disposing factor errors of diet are far inferior to alcoholic excess.

Malaria, by diminishing the resistance of the liver and producing a certain amount of hepatitis, disposes to hepatic abscess, and in a minor degree plays much the same part as alcoholism. Hepatic abscess, however, occurs in places where malaria is unknown. Malaria and dysentery occur so frequently in the same districts that malaria is often an antecedent condition of hepatic abscess. It is not, however, a true cause of hepatic suppuration.

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[1] Saundby and Miller. *Brit. Med. Journ.*, 1909, i, 771.

[2] Hilton Fagge's *Textbook*, edited by Pye-Smith, ii, 525, 4th ed.

[3] Sandwith. Quoted by Davidson, *System of Medicine* (Allbutt and Rolleston), 1907, ii, part ii, 591.

[4] Waring, E. J. *Hepatic Abscess*, p. 114, 1854.

[5] Megaw. *Ind. Med. Gaz.*, 1905, xl, 81.

Malta fever may precede, and dispose in a similar way to, hepatic abscess (Gabbi[1]).

Yellow fever may precede the development of an hepatic abscess, but this is probably merely accidental, and there is no reason to think that there is any direct relation between them; though no doubt after yellow fever the resistance of the liver is weakened and the organ more liable to infection.

Sex.—Single tropical abscess is much commoner in males than in females. This applies to adults, for in children the incidence is about equal in the two sexes.

In E. J. Waring's 300 Indian cases 291 were males and 9 females, or 30 to 1; Davidson gives a lower ratio of 7 to 1. In 13 fatal cases at St. George's Hospital 11 were males; in one of the females the abscess was probably connected with cholelithiasis.

The greater predominance of the male sex is due to various factors. First, the larger number of male Europeans resident in tropical climates must be taken into account, for Europeans are much more subject to abscess than natives. But even after allowing for this, males are more subject to abscess than women; for though dysentery affects European men and women equally, abscess seldom occurs in women. The factors which have been thought to increase the tendency of European males to abscess are: (i) Alcoholism, which disposes to hepatitis and favours infection by reducing the resistance of the organ; (ii) greater exposure to chills; and (iii) greater liability to blows in the hepatic region.

Age.—Hepatic abscess is a disease of adult life, and is rare in children and in old persons.

In 227 fatal cases tabulated by Waring,[2] 112, or 49·3 per cent, were between the ages of twenty and thirty. It must be remembered, however, that the cases were from the Indian army, which contained a very large number of men about this age. The oldest case met with by Waring was a pensioner aged seventy-two years, and the youngest in a girl aged fifteen years.

In young children large hepatic abscesses are rare; they may be due to dysentery, to worms in the bile-ducts, to trauma, or to appendicitis.

Legrand[3] collected 31 cases in children due to dysentery. Of Amberg's[4] 12 cases in children due to dysentery, only one (Slaughter's[5]) shewed motile

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[1] Gabbi. *Lavori dell' Istituto*, Messina, 1906, Fasc. i, 23.
[2] Waring, E. J. *Loc. cit.*, p. 112.
[3] Legrand. *Arch. de méd. des enfants*, Paris, 1906, ix, 129.
[4] Amberg. *Johns Hopkins Hosp. Bull.*, Balt., 1901, xii, 355.
[5] Slaughter. *Virginia Med. Monthly*, 1895–96, xxii, 722.

amoebae in the pus, though dead amoebae were seen in the pus of Gneftos's[1] case. Amoebic abscess is therefore very rare in children.

Niblock[2] records the case of a child aged eleven months who recovered after 18 oz. of pus had been removed from the abscess. Finizio[3] reports a fatal case of hepatic abscess subsequent to dysentery in a boy aged six years. In a boy aged nine years the abdomen was so distended that the condition was thought to be ascites (Hatch[4]).

Morbid Anatomy.—*Situation.*—Between 60 and 80 per cent of large single abscesses are in the right lobe of the liver. Occasionally a large abscess may occupy parts of both the right and left lobes.

Of E. J. Waring's cases, 67 per cent were in the right lobe; 6·6 per cent in the left lobe; and 14·5 per cent in both the right and the left lobe. In Rouis's[5] series 78·6 per cent were in the right lobe, 16·8 per cent in the left, and 4·6 per cent in the Spigelian lobe. From injection with methylene-blue, Sérégé[6] states that the blood from the stomach and spleen is carried into the left lobe, and that from the pancreas and intestines into the right lobe. This serves in part to explain the greater frequency of abscess in the right lobe. In addition, the right lobe being six times the size of the left naturally receives the larger share of blood. Rogers[7] suggests that the frequency of dysenteric ulceration in the ascending colon and hepatic flexure, which would favour the passage of amoebae across the peritoneum to the surface of the liver, may also account for the preponderance of abscess in the right lobe.

The abscess is often deeply embedded in the right lobe towards its posterior and upper border. When in this situation it may work its way forward and present anteriorly, but more often it projects upwards under the diaphragm. In the latter site the existence of an abscess is often very difficult to determine with certainty by percussion, as it may carry the diaphragm upwards, and if the lung is adherent to the chest wall there will be comparatively little dulness. In such cases skiagraphy is of great service, by shewing the position and amount of movement of the right leaflet of the diaphragm. The under surface of the right lobe of the liver close to the hepatic flexure of the colon is another favourite site of abscess. ~~According to Lafleur[8] and Rogers, it is probable that~~ In rare instances amoebae may pass through the walls of the colon and infect the surface of the liver ~~at this point~~ (Lafleur; Rogers.

When an abscess is near the surface of the liver and close to the suspensory ligament, it may rupture into the space between the two layers of that peritoneal ligament and form an abscess between the liver and diaphragm. This has been spoken of as a suprahepatic abscess by

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[1] Gneftos. *Deutsche med. Wchnschr.*, 1891, xvii, 853.

[2] Niblock. *Ind. Med. Gaz.*, 1911, xlvi, 137.

[3] Finizio. *Pediatria*, Napoli, 1896, iv, 340.

[4] Hatch. *Ind. Med. Gaz.*, 1898, xxxiii, 285.

[5] Rouis. *Recherches sur les suppurations endémiques*, 1860, Paris.

[6] Sérégé. Vide *Bull. et mém. Soc. méd. des hôp. de Paris*, 1901, 3. s., xviii, 393; *Journ. de méd. de Bordeaux*, May 25, June 1 and 8, 1902.

[7] Rogers, L. *Brit. Med. Journ.*, 1902, ii, 850.

[8] Lafleur. Allbutt's *System of Medicine*, 1897, iv, 156.

Cantlie,[1] who, however, considers that this form of abscess begins in the lymphatics in the ligament and not in the liver.

Number.—A large abscess may be the only focus of suppuration in the liver or it may be associated with one or more abscesses, usually smaller.

In E. J. Waring's 288 fatal cases 177, or 61·4 per cent, were solitary abscesses, and in 33, or 11 per cent, there were two abscesses. Davidson[2] gives 75 per cent as solitary, thus agreeing with Rouis, 11 per cent with two abscesses, and 14 per cent with more than two. In 509 necropsies tabulated by the Sanitary Commissioner with the Government of India there were 34 per cent of single and 66 per cent of multiple abscesses.[3] In 13 fatal cases at St. George's Hospital there was a single abscess in ten.

The smaller abscesses may be secondary and due to infection from the originally single abscess, or they may belong to a series of small abscesses which have united to form ~~an areolar~~ abscess. Two or even three large abscesses may be independent in origin. Godlee[4] believes that many of the cases of a second abscess following a previous one are due to the drainage-tube having been removed too soon, or to some diverticulum of the original abscess having been imperfectly explored and drained.

The *shape* of an abscess varies considerably; usually it is round, but it may be oval or irregular, and when due to the union of previously separate abscesses, may be branching ~~or areolar~~.

The *size* of a single abscess varies greatly. Not uncommonly it contains three-quarters of a pint of pus, but as much as 16 or even 19 pints have been recorded.

Morbid Appearances of the Liver.—The surface may shew recent inflammation and adherent fibrin over the abscess, or, when the process is of some duration, fibrous adhesions uniting it to the diaphragm, abdominal wall, or adjacent viscera. These are most frequent between the convexity of the right lobe and the diaphragm; the abscess may then project upwards under the dome of the diaphragm and give rise to comparatively little enlargement of the liver in a downward direction.

In its earliest stage a liver abscess appears as a pale, softened area, sometimes irregular in shape from the union of two or more such foci. This condition of commencing suppuration if aspirated may be recognised by finding cylinders of liver cells in the blood-stained material withdrawn, which, however, has no naked-eye resemblance to pus. The softened areas break down into recent abscess cavities which have ragged walls lined by necrosing liver tissue. Immediately around the spreading abscess the liver substance is softened, buff-coloured from swelling, degeneration, and necrosis of the liver cells and infiltration with polymorphonuclear leucocytes. The vessels may contain emboli of pyogenic cocci. As the

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[1] Cantlie, J. *Brit. Med. Journ.*, 1899, ii, 646.

[2] Davidson. *System of Medicine*, 1907, ii, part ii, 592.

[3] Report Sanitary Commissioner with Govern. of India, 1901.

[4] Godlee, R. J. *Med.-Chir. Trans.*, Lond., 1902, lxxxv, 119.

The formation of a single abscess by spread from the centre may suggest the union of previously separate abscesses (Vide Fig: 21)

inflammatory change spreads outwards it may set up suppurative thrombosis in the portal spaces and thus give rise to foci of suppuration in the neighbourhood, which may eventually open into the original abscess.

In an abscess of some standing there is lining of granulation tissue, which is ragged at first, and later becomes smooth. Outside this there is a fibrous capsule which runs into and invades the surrounding liver substance for a short distance. The internal lining of granulation tissue may have debris and fibrin adherent to it, and is often necrotic on the surface. The granulation tissue contains newly-formed vessels, and in its deeper layers developing fibrous tissue. In this way a capsule is formed, which in old abscesses is of considerable extent, and is formed of dense, well-formed fibrous tissue. This fibrous tissue invades the liver for a short distance and passes between the liver cells, which are flattened and often spindle-shaped, so as to suggest the erroneous view that they become transformed into fibrous tissue. The liver cells proliferate and give rise to columns of small cubical cells—the so-called new bile-ducts—and as a result the developing fibrous capsule around an abscess of some duration contains altered liver cells and pseudobile canaliculi. The capsule of old abscesses is often pigmented, and may be calcified. Occasionally suppurating foci are found outside the comparatively well-formed wall of a single abscess.

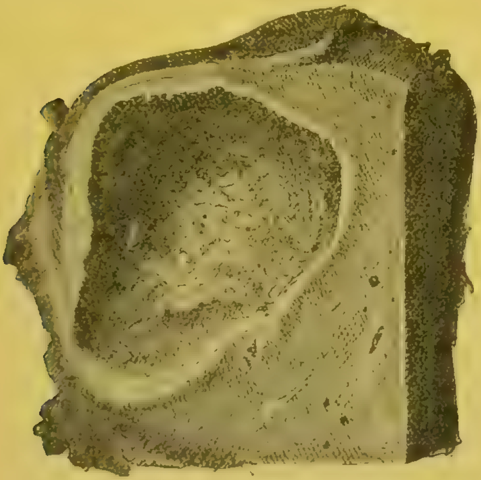

Fig. 20.—Single abscess with a definite capsule. A glass rod shews where it ruptured into the inferior vena cava. From a specimen (series ix, 171M) in St. George's Hospital Museum. (Drawn by L. Jones, M.S.)

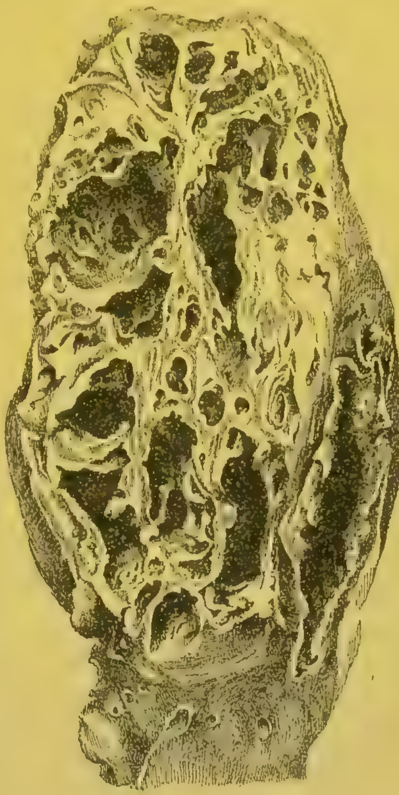

Fig. 21.—Section of the right lobe of the liver, shewing the union of a number of separate abscesses into an areolar abscess cavity.^ From a specimen (series ix, 171c) in St. George's Hospital. (Drawn by Dr. E. A. Wilson.)

[illegible]
^ which accor
to [illegible] is
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The *rest of the liver* is usually enlarged, and after evacuation of the abscess heavier than in health. In 132 cases tabulated by Waring this was the condition of the organ in 113. ~~The large size is chiefly due to parenchymatous inflammation and cloudy swelling, but, in addition, compensatory hypertrophy of the remainder of the liver occu~~rs. ∧ ~~In chronic cases in which there has been a fistula, increase in size of the liver may be due to lardaceous diseas~~e. There may, in addition, be pylephlebitic abscesses in the liver. In rare instances the liver substance may be bright yellow in colour and firm in consistence, resembling the appearances in phosphorus poisoning.

I met with this appearance in a case of a loculated liver abscess due to infection derived from the appendix which contained a pin.[1] In St. Bartholomew's Hospital Museum there is a specimen (2196D) of multiple abscesses with this appearance. E. J. Waring[2] in 113 fatal cases mentions one in which the liver had this aspect.

The scars of former abscesses that have been opened are sometimes seen. Abscess is very rare in a genuinely cirrhotic liver; in Waring's 113 cases the rest of the liver was cirrhotic in two.

In *amoebic abscesses* of the liver the histological appearances are so peculiar that special reference must be made to them. In small and recent abscesses there is a glairy, translucent fluid, which when removed leaves a shreddy, spongy wall. The wall is irregular from the remains of the portal canals, which are left by the necrosis and disintegration of the surrounding liver cells. The liver cells necrose probably as the result of the action of toxic substances manufactured by the amoebae, and then liquefy and disintegrate. Amoebae are found in the capillaries in the immediate neighbourhood of the abscess. There is a remarkable absence of leucocytic massing in the neighbourhood of the small abscesses ~~which is quite characteristic (Lafleur[3])~~. The process is one of colliquative necrosis ∧ rather than of inflammation. ~~Rogers,[4] however, who regards the small multiple amoebic abscesses as due to a mixed infection of amoebae and pyogenic cocci, figures small-celled infiltration in these minute abscess~~es.

hepatolysis /∧

Large chronic amoebic abscesses differ little from the appearances in other chronic hepatic abscesses. Amoebae are much less numerous than in the recent small amoebic abscesses, but are found in the same situation, viz. in the wall of the abscess cavity.

Contents of the Abscesses.—In large abscesses the character of the pus may vary very considerably; it may be white and creamy, yellow, brownish-red or chocolate colour, green; thick, mucoid, or even serous. The red colour is due to admixture with blood and the remains of the liver tissue. In amoebic abscess the contents of the smaller abscesses are

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[1] Rolleston. *Trans. Path. Soc.*, 1898, xlix, 106.

[2] Waring, E. J. *Abscess of the Liver*, p. 137, 1854.

[3] Lafleur. Allbutt's *System of Medicine*, 1897, iv, 158.

~~[4] Rogers, L. *Brit. Med. Journ.*, 1903, i, 1318~~.

Rogers states that in pure amoebic abscesses the liver parenchyma is normal; but when bacterial infection after operation has supervened, degenerative changes, amyloidosis or multiple abscesses may result. Before this [illegible] infection has occurred, compensatory hyperplasia accounts for much of the increased size.

(Lafleur)
due to pure amoebic infection which contrasts with the small-celled infiltration seen in small multiple abscesses due to a mixed amoebic and streptococcic infection

Rogers, L. *Trans. Med. Soc. London*, 1923, XLV, 130

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[illegible]
ar [illegible]

translucent, glairy, and do not flow easily. In larger amoebic abscesses the fluid is very viscid and does not resemble ordinary pus.

Microscopically the amoebic abscesses contain amoebae, which are naturally more numerous in the recent abscesses, necrosing liver cells, and red blood-corpuscles. Very few leucocytes are present, and the contents thus differ markedly from ordinary pus. Charcot-Leyden crystals have been found (Kruse and Pasquale[1]). Amoebic abscesses may be free from any pus-producing bacteria; Rogers says ~~this is so in~~ 80 per cent of the ~~cases;~~ they may contain various micro-organisms, such as streptococci, staphylococci, *Bacillus coli.* The pus is said to have a mawkish taste, and is not, as a rule, offensive. In abscesses on the under surface of the liver in contact with the bowel the contents may have a very faecal odour, due probably to secondary infection with members of the colon group. Da Costa[2] mentions an abscess of the liver secondary to appendicitis, which contained gas produced by bacteria.

Bacteriology.—Hepatic abscesses may be divided into (i) those which contain amoebae—called dysenteric by Kartulis; and (ii) the non-amoebic, due to micro-organisms, usually absorbed from the alimentary tract, and termed by Kartulis idiopathic.

Amoebae.—Since the discovery of amoebae in dysentery and in hepatic abscesses, some uncertainty has naturally arisen as to what proportion of the cases of tropical liver abscesses are due to this cause and to what extent amoebae are concerned in the production of suppuration.

Three species of amoebae occur as internal parasites of man; it is usually stated that the *Amoeba buccalis,* met with in patients with carious teeth, and the *Amoeba coli* Lösch occurring in the colon are harmless, and that the *Entamoeba histolytica* Schaudinn is the only one concerned in amoebic dysentery; but recent observations shew that this view is too sweeping (Greig and Wells[3]).

The causal relation of amoebae to the amoebic abscesses of the liver has given rise to a considerable amount of discussion, and the question is very far from being settled. The following views have been held.

(1) Kartulis,[4] who first recognised amoebae in the pus of liver abscesses, believed that they played an important accessory part in the production of abscesses, by conveying pus-producing micro-organisms to the liver and by rupturing the hepatic capillaries by their active movements; the production of pus not being due to amoebae, but to the pyogenetic micro-organisms.

(2) Kruse and Pasquale regarded the disintegration of the liver and abscess formation as due to a direct co-operation of the amoebae with micro-organisms.

(3) It has been suggested that the presence of amoebae in liver

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[1] Kruse und Pasquale. *Ztschr. f. Hyg.*, 1894, xvi, 1.

[2] Da Costa. *Modern Surgery*, p. 875, 1907.

[3] Greig and Wells. *Scientific Memoirs by Officers of the Medical and Sanitary Departments of the Government of India*, 1911, N.S., No. 47.

[4] Kartulis. *Centralbl. f. Bakt.*, 1887, ii, 745; *Virchows Arch.*, 1889, cxviii, 97.

abscesses is a secondary infection, and that they gain a footing in the liver only when it has been damaged by bacterial invasion (Sheldon Amos[1]). This view is supported by the absence of amoebae from some cerebral abscesses in cases in which they are found in the pus from the liver. Kelsch and Nimier[2] deny that amoebae are responsible for the production of liver abscess.

(4) Councilman and Lafleur[3] believe that the amoebae alone are responsible for the abscess, since in the smallest abscesses bacteria are absent while amoebae are plentiful. In larger abscesses bacteria are not numerous, and the lesions are not like those produced by bacteria. These lesions are largely necrotic, and are thought to be due to a soluble toxin manufactured by the amoebae. L. Rogers[4] endorsed Councilman and Lafleur's view that amoebae alone give rise to large hepatic abscesses, as he finds that at least two-thirds of amoebic abscesses are quite free from ordinary pyogenetic organisms~~, and in the view that amoebae may work their way from the bowel across the peritoneum to infect the surface of the liver~~.

When an abscess is opened, amoebae usually do not appear in the pus until some days have elapsed; the explanation of this is that amoebae are chiefly found in the walls of the abscess and not in its contents. By scraping the wall of the abscess amoebae may be found, though absent from the pus (Rogers[5]). In hepatic abscesses examined in England, Manson[6] found amoebae in considerably over 50 per cent.

Bacteria.—In non-amoebic cases bacteriological examination of the pus has given discordant results. In some instances the pus is sterile; this has been explained by supposing that in chronic cases the micro-organisms originally present have died out. Pus-producing micro-organisms have frequently been found, such as the *Staphylococcus pyogenes aureus*, *albus*, *citreus*, and streptococci. Typhoid bacilli have been found in pure culture (Cassuto[7]) or in combination with other micro-organisms. Ordinary pneumococci, Friedländer's pneumobacillus, *Bacillus pyocyaneus*, and the colon bacillus are also sometimes present. Eyre and Fawcett[8] described a case in which there was a pure culture of *Micrococcus melitensis*. Diplococci resembling gonococci were obtained from an hepatic abscess of a native Egyptian who denied gonorrhoea (Bousfield[9]). In an areolar abscess Hewitt[10] found *Bacillus mucosus capsulatus*.

Inflammation of the *pleura* is a very common occurrence in hepatic abscess; it may be due to a spread of infection along the lymphatics and may be dry, serous, or purulent. Pleurisy may give rise to universal

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[1] Sheldon Amos. *Journ. Path. and Bacteriol.*, 1902, viii, 346.
[2] Kelsch et Nimier. *Bull. Acad. de méd.*, Paris, 1900, xliii, 237.
[3] Councilman and Lafleur. *Johns Hopkins Hosp. Rep.*, 1891, ii, 395.
[4] Rogers, L. *Brit. Med. Journ.*, 1902, ii, 844; 1903, i, 1315; 1906, i, 1397.
[5] *Idem. Ibid.*, 1905, ii, 1291.
[6] Manson. *Tropical Diseases*, p. 456, 1903.
[7] Cassuto. *Thèse de Paris*, 1900.
[8] Eyre and Fawcett. *Guy's Hosp. Rep.*, 1905, lix, 206.
[9] Bousfield. *Journ. Roy. Army Med. Corps*, 1908, x, 80.
[10] Hewitt. *Johns Hopkins Hosp. Bull.*, Balt., 1909, xx, 77.

adhesions and so prevent the abscess rupturing into the pleura, but thus favour its rupture into the lung. An empyema due to this course may, as in Duplant's case, be encysted between the lobes of the lung. Rupture or direct leakage of an abscess into the pleura will give rise to a large empyema.

Lungs.—The lower lobe of the right lung may be compressed by the upward pressure of the liver and diaphragm, or by a pleural effusion; or if it is adherent to the diaphragm it may become inflamed from direct extension of inflammation from the abscess through the diaphragm, or the abscess may burst into the lung. In cases in which the abscess has leaked into the hepatic veins or into the inferior vena cava there may be pyaemic abscesses in the lungs. The rupture of an hepatic abscess into the lung will be referred to on p. 141.

Clinical Picture.—*Symptoms.*—Latency; onset; fever; sweating; rigors; pain; shoulder pain; tenderness; cough; digestive disturbances; arthritis; nervous disorders.

There is very great variation both in the individual symptoms and also in their association; this is shewn by the number of conditions for which hepatic abscess has been mistaken (*vide* p. 146). Very frequently the symptoms suggest rather than definitely point to hepatic abscess.

Latency.—An abscess may remain perfectly latent and only be found at death; according to Rouis, this occurs in 13 per cent. In some cases there are no definite symptoms or signs until the abscess ruptures and rapidly brings about a fatal result.

The onset of symptoms is gradual, as a rule, and is ushered in by malaise, languor, general debility, and the signs and symptoms of acute congestion of the liver and acute hepatitis (*vide* p. 115). Indefinite malaise may precede by many weeks any definite evidence of abscess. In some instances there is an acute onset with shivering or a rigor, so as to imitate pneumonia of the right lower lobe.

Fever is perhaps the most constant sign of hepatic abscess. The temperature varies; it may be continuous at first, then remittent, and in the later stages intermittent. It may closely imitate malaria, or be extremely irregular. In chronic cases the temperature may be normal, and in severe and advanced cases it may be low and even subnormal.[1] In exceptional instances there may be little or no fever, even with a large and recent abscess. There may be practically no pyrexia from start to finish.

Moir[2] records an abscess containing 70 ounces of pus, in which the temperature ranged between subnormal and 99° F.

Sweating.—Nocturnal sweats may be very profuse. Perspiration often occurs during the day when the patient drops off to sleep. In fact, the sweating is more related to sleep than to the night hours, and is chiefly nocturnal because sleep is more general then.

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[1] Smith, Johnson. *Brit. Med. Journ.*, 1900, ii, 550.

[2] Moir, D. M. *Ind. Med. Gaz.*, 1897, xxxii, 218.

Rigors are often met with in the course of the disease. In some cases there is a single rigor at the onset or early in the course of the disease; in others there are frequent rigors; in some instances they do not appear until near the end of the case. A feeling of chilliness often accompanies the evening rise of temperature.

Pain over the liver and a sense of fulness in the right hypochondrium are very commonly present. Though pain may be present from the start of suppuration, it is much more in evidence when the abscess is large and approaching the capsule of the liver, than early in the process or when the abscess is deeply situated. The pain may be constant and dull, probably from increased tension inside the liver; this is usually early in the disease; or intermittent and sharp and stabbing, and, like pleuritic pain, brought on by respiratory movements. It may be actually due to pleurisy or to inflammation of the capsule of the liver, and is then rather a late symptom. Pain on swallowing, due to disturbance of the liver by food entering the stomach, is mentioned by Manson.[1]

Shoulder Pain.—In abscess, as in some other affections of the liver, pain may be referred to the shoulder, and may be felt over the scapula or at the tip of the acromion process. According to Manson, it occurs in about one-sixth of the cases. It has been suggested that this localisation may depend on the communication between the nerve to the subclavius muscle and the phrenic nerve. It is most frequently present when the abscess is in the upper part of the right lobe. In abscesses in the left lobe the pain may be referred to the left shoulder. Very rarely the pain is bilateral (E. J. Waring [2]).

Tenderness.—On deep palpation or percussion tenderness of the liver is generally elicited, and is perhaps the least unsatisfactory sign of hepatic abscess.[3] This localised tender spot is a useful guide to the position of the abscess. Occasionally vomiting is constantly induced by pressure on one spot (Smits [4]).

Cough.—In hepatic abscess there is often a spasmodic hacking cough, which is usually worse at night, and may be ineffectual or unaccompanied by sputum. It may be due to irritation of the pleura over the diaphragm, to actual pleurisy, or possibly merely to reflex irritation from the liver. It is not peculiar to liver abscess. When the abscess discharges through the lung, severe cough comes on and expectoration may be copious. As much as 4 pounds of pus has been brought up in twenty-four hours (Waring [5]). The colour of the expectorated matters differs greatly in different cases; it may be mixed with bile. In amoebic abscesses amoebae may be found in the sputum before the abscess has burst into the lung.

Digestive Symptoms.—Vomiting appears to be more frequent when the

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[1] Manson, P. *Tropical Diseases*, p. 353, ed. i, 1898, London.

[2] Waring, E. J. *Abscess of the Liver*, p. 147, 1854.

[3] Smith, Johnson. *Brit. Med. Journ.*, 1900, ii, 550.

[4] Smits. *Arch. f. klin. Chir.*, 1900, lxi, 173.

[5] Waring. *Loc. cit.*, p. 153.

[illegible] up [illegible]
the lower border

and raising the diaphragm.

or even haematomesis; Brau and
Nogue regard these haemorrhages
as due to hepatic insufficiency and
of very grave prognostic significance

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abscess is in the left lobe or when there is an abscess in the left lobe together with one in the right. In very rare instances vomiting may be due to the abscess pressing on the pylorus and leading to pyloric obstruction. Maclean[1] described pyloric obstruction due to hepatic abscess. Appetite is poor or completely lost, as a rule, but sometimes it is well preserved. Flatulence may be troublesome.

There may be constipation, diarrhoea, or the two may alternate. Absorption from the abscess may give rise to ulceration of the intestine and diarrhoea. A recrudescence of a former dysentery is not uncommonly described. When the abscess bursts into the alimentary tract, large quantities of pus may appear in the stools. In rare cases there may be considerable haemorrhage from the bowel.[2]

Captain Weston, R.A.M.C., has told me of a case in which there were profuse haemorrhages from the bowel apparently due to an abscess in the quadrate lobe of the liver pressing on the portal vein.

Arthritis, etc.—Swelling and painful enlargement of the joints may occur, just as in dysentery. They may be pyaemic, but are probably usually toxic; at any rate, they tend to disappear when the abscess is opened or discharges. Girard[3] observed clubbing of the fingers in a case of hepatic abscess free from pulmonary lesions. Possibly this change was toxic.

Nervous Symptoms.—As in most hepatic diseases, there is a good deal of irritability or mental depression, which may even become so marked as to pass into melancholia. Insomnia is very common. The mental disturbance may be referred to the action on the cerebral cortex of poisons absorbed from the liver abscess or from the alimentary tract, the liver being unable to deal with them. Cerebral symptoms in rare instances depend on pyaemic abscesses in the brain or on meningitis. In the terminal stages delirium or, in rare instances, convulsions may occur.

Physical Signs. — Wasting; decubitus; facial aspect; jaundice; pulse; blood; respiratory system; abdominal signs—painful succussion, skiagraphy, rigidity of right rectus muscle, ballottement, hepatic dulness, friction; oedema of feet; urine.

Wasting is progressive and may be very considerable; its degree necessarily depends on the duration and intensity of the disease and the size of the abscess. In rare instances weight may actually be put on while an abscess is maturing (Osler[4]).

Decubitus, or the position assumed by the patient, is generally dorsal or right-sided, as being that in which there is greatest freedom from pain. When the patient lies on his back, the chest is raised and the knees are somewhat flexed. When he turns on to the left side, the liver tends to fall away from the wall of the abdomen on the right side, and pain is frequently felt. This is probably due to stretching of adhesions or to the separation of the inflamed capsule of the liver from the parietal peritoneum. On the other hand, the patient is sometimes more comfortable on the left side. The differences in the position assumed by the patient may depend on the increased size of the liver and on the presence of adhesions. Thus, when the liver is greatly enlarged the right-sided position is most comfortable; but with peritoneal adhesions in certain situations, the right-sided position may give rise to tension on them and so to pain.

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[1] Maclean. *Brit. Med. Journ.*, 1874, ii, 138.

[2] Hassler. *Deutsche med. Wchnschr.*, 1902, xxviii, 47.

[3] Girard. *Compt. rend. Soc. Biol.*, Paris, 1903, liv, 106.

[4] Osler. *Med. News*, N.Y., 1902, lxxx, 673.

Facial Aspect.—The complexion is pale or sallow with a muddy tint, and often slightly icteric. The conjunctivae if not jaundiced are often of a waxy, white colour. The expression is anxious and worried, the eyes are often sunken, and the general aspect, which is more easily recognised than described, may be peculiarly suggestive of the disease. The skin in the early stages may be hot and dry, but later it becomes moist and clammy.

A remarkable diffuse cyanosis, for which there was no satisfactory explanation, was noticed by Osler in one case.

The tongue is usually furred along the centre with a red tip. A smooth red tongue with fissures running in all directions is not uncommon. Brun,[1] indeed, considers a bright red and absolutely dry tongue to be pathognomonic. A strong hepatic odour from the patient is described by Hatch[2]; probably it is present only when the abscess is of considerable size.

Jaundice is very rarely deep; a slight degree, probably due to local catarrh of the ~~intrahepatic~~ ducts near the abscess, and tingeing the conjunctivae, ~~are~~ is sometimes present. In exceptional cases an abscess may press on the extrahepatic ducts in the portal fissure (Moir[3]).

Definite jaundice was seen in 58 out of 375 cases, or in 16 per cent (Thierfelder[4]).

The pulse is of low tension, full, and somewhat quickened—80 to 100. Exceptionally it is slow.

The blood may shew leucocytosis; and this, when present, is of great service in distinguishing hepatic abscess from malaria or typhoid fever. The existence of considerable leucocytosis—30,000 to 50,000 per cubic millimeter—has led to the detection of abscess in patients formerly the subjects of dysentery (Boinet[5]) or thought to have malaria (Rogers[6]). When leucocytosis is present, it is said to be constant (Boinet), and not to appear and disappear as it does in intermittent hepatic fever. The

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[1] Brun. *Rev. de méd.*, Paris, 1904, xxiv, 396.
[2] Hatch. *Brit. Med. Journ.*, 1900, ii, 1374.
[3] Moir. *Ind. Med. Gaz.*, 1902, xxxvii, 391.
[4] v. Ziemssen's *Cyclopaedia of Practical Medicine*, 1880, ix, 126.
[5] Boinet. *Compt. rend. Soc. Biol.*, Paris, 1900, lii, 1089.
[6] Rogers, L. *Brit. Med. Journ.*, 1902, i, 831; ii, 844; 1905, ii, 1291.

differential count is usually said to give a high percentage (75 to 80) of polymorphonuclears; according to Marchoux[1] and Rogers[2] this is true of abscesses due to bacterial infection, but not of amoebic abscesses in which there is increase of the mononuclears. Leucocytosis, though highly suggestive, is not constantly found in abscess. When absorption is prevented by a thick capsule there may be no leucocytosis. It has been thought that leucocytosis is most marked in small, deeply seated abscesses, and less in large abscesses bulging on the surface of the liver (Rogers).

It was absent in 3 out of 5 cases recorded by Osler,[3] and in 6 cases mentioned by Charles[4]; Rispal[5] failed to find more than a slight leucocytosis (15,000 per cubic millimeter) in 3 cases.

Iodophilia, or the iodine reaction in the polymorphonuclears, occurs unless the abscess is so thick-walled as to prevent absorption (Da Costa[6]). Blood-cultures are said to be negative by Libman[7]; but in bacterial cases the blood may shew the corresponding micro-organism (Simon[8]).

Respiration is shallow and chiefly thoracic, since movement of the abdomen may cause pain. The rhythm is more rapid than normal, both because of the fever and on account of the shallow respirations. In cases of amoebic abscess the organisms may be found in the expectoration.

Lung Signs.—Compression of the lower lobe of the right lung, as shewn by crepitations and dulness, is common. When inflammation has spread through the diaphragm to the pleura, friction may be heard. Hepatic abscess may therefore imitate pneumonia or empyema at the right base; right-sided pleurisy in a patient who has had dysentery should always suggest the possibility of an abscess of the liver. The appearance of pleuritic friction in a case of suspected hepatic abscess is a sign that the inflammatory process is spreading to the thorax, and is therefore a signal for operative interference, so as to prevent rupture of the abscess into the pleura or into the lung. But in some cases of acute hepatitis which have been fruitlessly explored and have recovered, friction at the base of the right lung has been present.

Hepatic abscess may cause an empyema in several ways. The abscess may burst directly into the pleura, or the infection may spread through the diaphragm, or pus may pass into the pulmonary artery and set up abscess formation in the lung with the secondary production of an empyema. An empyema may form between the lobes of the lung and be very difficult to evacuate.

Rupture of an hepatic abscess into the lung may give rise to the signs of a cavity in the lower lobe, which, according to Godlee,[1] are usually below and to the inner side of the right nipple, but may be present posteriorly.

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[1] Marchoux. *Brit. Med. Journ.*, 1908, ii, 1252.
[2] Rogers, L. *Med.-Chir. Trans.*, London, 1907, xc, 163.
[3] Osler. *Med. News*, N.Y., 1902, lxxx, 673.
[4] Charles. *Brit. Med. Journ.*, 1908, ii, 1235.
[5] Rispal. *Compt. rend. Soc. Biol.*, Paris, 1901, liii, 862 .
[6] Da Costa. *Therap. Gaz.*, Detroit, 1906, xxii, 676.
[7] Libman. *Am. Journ. Med. Sc.*, Phila., 1908, cxxxvi, 518.
[8] Simon. *Clinical Diagnosis*, p. 641, 1911.

The heart's apex may be displaced upwards, into the fourth space, especially when the abscess is in the left lobe of the liver.

The abdomen is full and somewhat distended; sometimes the distension is general, as if from tympanites. There is often definite fulness or bulging in the right hypochondrium or epigastrium, and sometimes a rounded projection in the position of, or in connexion with, the liver may be seen to move with respiration. In some cases fluctuation can be made out over the tumour. An abscess near the middle line of the body may shew transmitted impulsation from the aorta.

Carpenter[2] describes a painful swelling the size of a fetal head pulsating visibly, but not expansile; operation proved that it was an hepatic abscess containing two pints of pus.

Bulging of the chest on the right side as compared with the left, and in some cases protrusion and widening of the intercostal spaces, may be seen. Redness of the skin or localised oedema indicates that an abscess is in close contact with the affected cutaneous area. Though a very valuable sign, oedema over the abscess is only present in about 5 per cent of the cases (Stevenson[3]). A very large abscess may fill the greater part of the abdomen, and has given rise to a diagnosis of ascites. Much ascites is extremely rare. Bradshaw[4] records a case in which ascites appeared to be due to the pressure exerted on the portal vein by an abscess in the Spigelian lobe. It is possible that it may be induced by puncture of the liver with subsequent escape of blood into the peritoneal cavity; I have seen one case suggesting this sequence of events.

Painful Succussion.—If the patient is shaken, much in the same manner as in obtaining "Hippocratic succussion" in pyopneumothorax, pain is felt in the liver and may spread to the right shoulder, the pit of the stomach, across the abdomen, or to the right iliac fossa, according to the situation of the abscess. Malbot[5] considers this a certain sign of abscess.

Skiagraphy.—In health the shadow cast on the screen shews that the right leaflet of the diaphragm is 1½ inches above the level of the left leaflet, and moves with respiration. In cases of abscess it is displaced upwards and does not move. ~~Dr. Herringham has told me of a large hepatic abscess which displaced the diaphragm upwards, but did not prevent it from moving freely with respiration.~~ The position of the abscess may be shewn by increased density of the shadow.

Rigidity of the upper part of the right rectus muscle may certainly occur when an abscess is present, but it is far from constant; moreover, it is

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[1] Godlee. *Med.-Chir. Trans.*, Lond., 1902, lxxxv, 119.

[2] Carpenter. *Brit. Med. Journ.*, 1899, ii, 208.

[3] Stevenson. *Lancet*, 1898, i, 1428.

[4] Bradshaw. *Ibid.*, 1908, i, 146.

[5] Malbot. *Arch. gén. de méd.*, 1899, clxxxiv, 172.

There may be a dome shaped
elevation of the diaphragm

met with in other conditions, such as inflammation of the gall-bladder, disease of the pylorus, duodenal ulcer, or inflammation of the muscle itself, and therefore is not a sign of any very special value. The rigidity of the rectus may considerably interfere with the examination of the liver. Dalton[1] refers to a case in which the rigidity did not relax under an anaesthetic.

Percussion, besides eliciting tenderness and causing pain, may convey to the examiner a sensation of *ballottement,* as if he were percussing a thick-walled elastic bag filled with air.[2]

Hepatic Dulness.—The area of hepatic dulness is increased both in an upward and in a downward direction. Though this is the rule, it is not without exception, and in some instances the underlying colon may mask the dulness which a collection of pus would naturally give rise to. The dulness in extreme cases may reach as high as the second rib (Waring[3]). As the abscess is usually in the right lobe, the enlargement is mainly on that side, and since the abscess is most frequently near the convexity of the liver, the enlargement is chiefly in an upward direction and not downwards, as in cirrhosis and in lardaceous and malignant disease. When an abscess is in the posterior and upper part of the right lobe there may be no hepatic enlargement below the costal arch. When the abscess is deeply situated, the whole lobe is expanded and enlarged, but later, when the abscess reaches the surface, it may give rise to a definite projection from the outline of the organ.

Friction due to perihepatitis, set up by the approach of the abscess to the surface of the organ, may be heard or sometimes even felt by the hand. There is commonly friction over the base of the right lung, due to pleurisy set up by the spread of infection along the lymphatics of the diaphragm.

Fine crepitation, like that obtained by pressing on frozen snow, may be heard over the liver; it occurs with inspiration and expiration. Bertrand regarded it as due to perihepatitis, but Hassler and Boisson have heard it in cases in which laparotomy shewed that there was no perihepatitis, and believe that it is produced in the softened and oedematous liver substance around the abscess.

The *spleen* is rarely enlarged. This is of importance in distinguishing abscess from malaria.

Oedema of the feet often occurs late in the course of the disease.

The urine is high-coloured and may be of a high specific gravity, and loaded with urates. When considerable destruction of the hepatic substance has taken place, the amount of urea may be diminished and the ammonia increased. According to Axisa[4] the diminution on the amount of urea runs parallel with the size of the abscess. Indican may occur. Albuminuria is sometimes present. From absorption of pus albumose may be found in the urine; but it has been absent in the cases I have seen.

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[1] Dalton, N. *King's Coll. Hosp. Rep.*, 1896, ii, 25.
[2] Hassler et Boisson. *Rev. de méd.*, Paris, 1896, xvi, 785.
[3] Waring, E. J. *Abscess of the Liver*, p. 149, 1854.
[4] Axisa. *Zentralbl. f. inn. Med.*, 1905, xxvi, 929.

In a case of abscess following dysentery crystals of leucine, tyrosine, and cholesterol were present in the urine for ten weeks; on opening the abscess the urine became normal.[1] Crystals resembling tyrosine, but really composed of a soap of lime and magnesium, were found in one case by Pfahler.[2]

Course and Duration.—The abscess may remain latent until it ruptures and gives rise to acute symptoms or is revealed by a discharge of pus from the alimentary or respiratory tract. It is difficult to say when hepatitis passes into suppuration, and therefore to estimate precisely the duration of an abscess in the liver. An abscess may run its course in three weeks, or if it gives rise to a fistula, especially in connexion with the lungs, may drag on for many months.

Termination.—If not operated upon, an hepatic abscess may either remain intact or may rupture into some neighbouring cavity or viscus. An abscess which remains intact may in rare instances tend to dry up and become shut off and encysted, giving rise to a caseous mass surrounded by a fibrous capsule. How often this actually occurs it is difficult to say, inasmuch as an abscess cannot be certainly diagnosed until it has reached a considerable size, and would then be operated upon.

In 25 cases in which recovery took place, there were fair grounds for believing that the abscess underwent this spontaneous cure in two (E. J. Waring[3]).

Possibly this spontaneous cure is even less frequent than is sometimes thought, since some so-called "receding" abscesses found post-mortem may in reality have been gummas. If the abscess remains intact and does not discharge its contents spontaneously, it usually leads to death.

In 300 fatal cases tabulated by E. J. Waring, 48, or 16 per cent, were operated upon, while 169, or 56 per cent, died with the abscess intact.

Though the abscess practically remains intact, it may give rise to secondary abscesses in the lungs and to general pyaemia. Death may be due to detachment of a thrombus formed in one of the hepatic veins and pulmonary embolism. In some cases the destructive suppurative change in the lungs may cause fatal haemoptysis. As a rule, the fatal result is due to septicaemia and exhaustion. Diarrhoea or an apparent recurrence of dysentery may carry the patient off. In rare cases death may be directly due to intestinal haemorrhage (Hassler,[4] Duplant[5]).

Rupture.—If undetected or not operated upon, an hepatic abscess may rupture internally, or in very rare instances on the surface of the body.

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[1] *Lancet*, 190[illegible], ii, 1729.
[2] Pfahler. *New York Med. Journ.*, 1902, lxxv, 270.
[3] Waring, E. J. *Abscess of the Liver*, p. 195, 1854.
[4] Hassler. *Deutsche med. Wchnschr.*, 1902, xxviii, 47.
[5] Duplant. *Lyon méd.*, 1902, xcviii, 109.

The percentage of cases in which rupture occurs depends on the diagnosis and on the frequency with which operative measures are undertaken. In 563 cases of hepatic abscess collected by Cyr, 83, or 15 per cent, were operated upon, and rupture occurred in 159, or 28·6 per cent. But interesting examples of perforation of hepatic abscess are more likely to be put on record than more commonplace cases. More than half the cases of perforation, as will be seen from Cyr's and Thierfelder's tables, were in an upward direction—through the diaphragm.

	Cyr,[1] 159 Cases.		Thierfelder,[2] 170 Cases.	
Lung	59	57 per cent.	74	59 per cent.
Pleura	31		26	
Pericardium . . .	1		4	
Peritoneum . . .	39		23	
Stomach . . .	8		13	
Intestines . . .	13		32	
Kidney . . .	2		1	
Inferior vena cava . .	3			
Bile passages . .	4			
Externally . . .	2			

In both these series perforation occurred in two positions in three cases, so that the numbers of the perforations exceed the number of cases.

The various situations in which rupture occurs may conveniently be considered seriatim under the following heads: rupture into—(1) Lung, (2) Pleura, (3) Pericardium, (4) Peritoneum, (5) Stomach and duodenum, (6) Intestines, (7) Gall-bladder and bile-ducts, (8) Kidney, (9) Hepatic veins and Inferior Vena Cava, (10) Portal Vein, (11) On the surface of the body.

(1) *Rupture into the Lung.*—This is the most frequent method by which an hepatic abscess spontaneously discharges. In an overwhelming majority of cases it is the right lung into which the abscess opens. Before the abscess can extend into the lung the liver must become adherent to the diaphragm, and the diaphragm to the base of the lung. The inflammatory processes necessarily cause pain and usually signs of basal pleurisy and cough. Inflammation spreads into the lung and consolidation with suppuration ensues; the communication between the abscess in the liver and that in the lower lobe of the right lung is usually somewhat small, like a shirt-stud abscess, and may be missed; in some instances no naked-eye perforation of the diaphragm is forthcoming. In rare cases there may be a small collection of pus between the diaphragm and lung as well as an abscess in the lower lobe of the lung. The formation of an abscess in the lower lobe is, according to Lafleur, due to an extension of inflammation and infection by continuity from the liver, and not haematogenous and due to embolism through the pulmonary artery. On the other hand, amoebae have been found in the small branches of the pulmonary

[1] Cyr. *Traité pratique des maladies du foie*, 1887.

[2] Thierfelder. *Cyclopaedia of the Practice of Medicine*, v. Ziemssen, 1880, ix, 138.

artery, so that haematogenous infection is quite conceivable. The suppurative process in the lower lobe spreads into the surrounding pulmonary tissue and tends to produce fistulous passages in the lung which as a result becomes much disorganised. The abscess discharges into one of the bronchi in the lower lobe of the lung and characteristic sputum mixed with blood is coughed up. The sputum contains pus, blood, and often fragments of hepatic and pulmonary tissue, fat globules, micro-organisms, and amoebae. Occasionally a fistulous communication between the bronchi and a bile-duct in the liver may be established and bile may be brought up from the lungs.

The pulmonary abscess may progress, and, indeed, prove fatal from exhaustion or haemoptysis, when the original hepatic abscess has undergone complete cure. Since the pus is like that brought up from the liver and may contain amoebae, the expectoration of chocolate-coloured pus does not prove that the original liver abscess has not healed (Godlee[1]). Lafleur[2] gives a very unfavorable prognosis in rupture of amoebic abscess of the liver into the lung. Other writers take a very different view; Decastro,[3] for example, estimating that recovery occurs in 76 per cent of the cases.

(2) *Rupture into the pleura*, though much the same process, is less frequent than rupture into the lung. This depends on the fact that the abscess as it advances through the diaphragm tends to set up adhesive pleurisy and so glues the lower lobe of the lung to the diaphragm. When the abscess opens into the pleura, the signs are those of an empyema. The effusion may reach a very large size with great rapidity and seriously endanger life.

(3) *Rupture into the Pericardium.*—Rupture into the pericardium is very rare, since this is likely to occur only when the abscess is in the left lobe of the liver—a rare situation.

Waring[4] quotes 6 recorded cases and I have notes of 13 others, making 19 in all. In Bentley's[5] case there were four pints of sero-purulent fluid in the pericardium which communicated with an abscess in the left lobe of the liver. Rupture of an hepatic abscess which also communicated with the intestine has been known to set up a gaseous purulent pericarditis (Wilks[6]).

An abscess in the left lobe may leak on to the surface of the liver and set up a subphrenic abscess which may subsequently perforate into the pericardium (Tolot[7]). The clinical evidence of rupture into the pericardium is severe pain, dyspnoea, and the rapid development of a pericardial effusion. Death usually, but not invariably, follows very soon after this accident.

[1] Godlee. *Med.-Chir. Trans.*, Lond., 1902, lxxx, 119.
[2] Lafleur. Allbutt's *System of Medicine*, 1897, iv, 168.
[3] Decastro. *Des abcès du foie des pays chauds*, Paris, 1870.
[4] Waring, E. J. *Abscess of the Liver in the East Indies*, p. 133, 1854.
[5] Bentley. *Trans. Path. Soc.*, 1850, ii, 70.
[6] Wilks. *Lectures on Pathological Anatomy*, p. 108, ed. iii, 1889.
[7] Tolot. *Lyon méd.*, 1902, xcviii, 51.

(4) *Rupture into the Peritoneum.*—This is comparatively common. It may occur into the general peritoneal cavity, or may give rise to a localised intraperitoneal abscess which may subsequently burst into the general cavity of the peritoneum, or may discharge into the bowel or even externally. Rupture into the general peritoneum may be due to trauma or to muscular exertion, and the danger of leakage must always be borne in mind when aspiration with a trocar through the abdominal wall is contemplated. Perforation into the general peritoneal cavity is more likely to follow sudden exertion or trauma than to occur spontaneously, as an abscess pointing on the surface of the liver tends to set up local peritonitis and adhesions before actually rupturing. Rupture into the general cavity of the peritoneum causes severe collapse, and if the patient survives long enough, acute peritonitis, which is nearly always fatal. In cases in which the existence of an hepatic abscess has not been recognised, the sudden onset of acute symptoms may imitate perforation of an intestinal ulcer in ambulatory typhoid fever.

A soldier, aged twenty-seven, who had contracted dysentery in the Transvaal War of 1900, was admitted moribund into St. George's Hospital. The necropsy disclosed several pints of turbid fluid in the peritoneal cavity and general peritonitis due to leakage from an abscess in the left lobe of the liver. The ruptured abscess was thin-walled and quite small, it communicated with a large abscess the size of a fetus' head, which occupied the whole of the left lobe of the liver, and had walls composed of glistening fibrous tissue about ½ inch thick. The right lobe of the liver was greatly scarred from cicatrices, but did not contain any gumma; it shewed early lardaceous change. The portal vein was thrombosed; there had probably been ascites before the onset of acute peritonitis.

Hulke[1] recorded recovery from rupture of an hepatic abscess into the general peritoneal cavity which was flushed out some twenty-four hours after rupture occurred. In this patient an hepatic abscess had previously ruptured through the lung, and rather more than a year after recovery from the operation for rupture into the peritoneum, a third abscess in the liver was successfully operated upon.

(5) When rupture occurs into the *stomach or duodenum* pus is vomited, sometimes in very considerable quantities, and is passed by the bowel, but from the distance which it has to travel the pus may not be detected in the faeces. After rupture the size of the liver may perceptibly diminish, and in some instances a tympanitic note has resulted from entrance of air into the abscess cavity. Rupture into the stomach is preceded by dyspepsia and discomfort, and accompanied by vomiting and severe epigastric pain.

(6) *Rupture into the Intestines.*—An hepatic abscess very seldom opens into any part of the small intestine except the duodenum. Rupture into the colon is not uncommon; it is accompanied by colicky pain and collapse, and is followed by the passage of pus by the bowel.

(7) *Rupture into the gall-bladder or bile-ducts* is very rare. The pus may pass down the common bile-duct into the duodenum and so imitate

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[1] Hulke. *Med.-Chir. Trans.*, 1893, lxxvi, 81.

rupture of an abscess into the bowel. Symptoms of biliary colic have been noted.

(8) *Rupture into the pelvis of the right kidney* is very rare. H. J. Waring[1] estimates the recorded cases at about 10.

(9) *Rupture into the Hepatic Veins and Inferior Vena Cava.*—Since the hepatic veins are not surrounded by any fibrous sheath at all comparable to Glisson's capsule around the portal canals, an hepatic abscess is more likely to leak or rupture into them than into the branches of the portal vein. The abscess may burst into one of the hepatic veins quite close to the inferior vena cava. In a few cases hepatic abscess has perforated directly into the inferior vena cava and not into the hepatic veins.

Cyr's list contains 3 cases. ~~Flexner[2] (1897) described 2 cases and Novis 1.[3]~~ In a case in St. George's Hospital (Fig. 20) the abscess occupied a third of the liver and had set up parietal thrombosis in the inferior vena cava. The pulmonary artery contained firm thrombi and there was pneumonic consolidation of the right lower lobe.

When the abscess begins to penetrate the wall of the hepatic veins or inferior vena cava parietal thrombosis may be set up, and thus for a time prevent the free entry of pus into the circulation, but eventually pus passes into the blood-stream and may give rise to secondary abscesses in the lungs. Detachment of a clot may cause pulmonary embolism.

(10) *Rupture into the portal vein* is extremely rare. Occasionally the abscess is in contact with the wall of the vein and sets up thrombosis in the vein which would prevent the contents of the abscess from passing into the vein.

(11) *Rupture Externally on to the Surface of the Body* ~~—A liver abscess~~ is very seldom allowed to ~~rupture~~ occur spontaneously ~~on the surface of the body~~. Most frequently the abscess points anteriorly, but occasionally it presents in the loin.

Complications.—Apart from manifest rupture hepatic abscess may give rise to secondary inflammation and suppuration, especially in the thorax. The spread of inflammation through the diaphragm may cause a serous pleurisy on the right side. Infection may travel through the diaphragm and set up an empyema on the right side, which may be encysted between the lobes of the lung, as in Duplant's[4] case. Infection may spread through the diaphragm, produce pleurisy and adhesions between the base of the lung and the diaphragm, and then extend into the substance of the lower lobe, setting up pneumonic consolidation, in the middle of which an irregular abscess may develop. Rupture into the lung and pleura and broncho-biliary fistula have already been described.

Cerebral Abscess.—~~Jacob[5] collected 15 cases~~; ~~in one group~~ the cerebral abscess contained amoebae ~~in the other~~ and those in which this was not decided.

Of this category Armstrong says that there are only 10 on record!

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[1] Waring, H. J. *Diseases of the Liver*, p. 98, 1897, Edin. and London.
[2] Flexner. *Amer. Journ. Med. Sc.*, 1897, cxiii, 553.
[3] Novis. *Brit. Med. Journ.*, 1911, ii, 1408.
[4] Duplant. *Lyon méd.*, 1902, xcviii, 109.
~~[5] Jacob. *Rev. de chir.*, Paris, 1911, xliv, 549.~~

Mc Knight in 1928 collected 12 cases.

A) Legrand collected 43 cases accompanying or following liver abscess; and Armstrong brought the number up to 49; Jacob divided the cases into (1) those in which

Mc Knight, R. B. Journ. Am. Med. Assoc., 1928, xc, 1929

Legrand. *Arch. prov. de chir.*, Par., 1912, xxi, 1.

Armstrong, F. L. *Journ. Trop. Med. and Hyg.*, London, 1919, xxii, 69

Novis[1] refers to 5 cases seen at Bombay within 6 months. The cerebral abscess may be derived from an abscess in the lungs; in Godlee's[2] case the hepatic abscess had healed, but there were abscesses in the lungs.

As a result of firm adhesions, associated with an old amoebic abscess in the left lobe of the liver, fatal *strangulation of the small intestine* has been recorded (Rogers[3]). Adhesions about the portal fissure, in cases in which an abscess has been successfully operated upon, may compress and exert traction on the bile-ducts giving rise to jaundice, or on the colon or pylorus, producing kinking and dilatation of the stomach (Godlee[4]).

Thrombosis of the inferior vena cava is a very rare complication; a parietal thrombus may form when an abscess bursts into the inferior vena cava; ~~which~~ also is very rare. ; this

Pheasants[5] ~~has~~ collected 8 cases of thrombosis, and Rixford[6] ~~has~~ recorded obliteration of the inferior vena cava. In a case of hepatic abscess with extreme marasmus under the care of my colleague, Dr. Ewart, there was extensive thrombosis of the inferior vena cava, which appeared to have spread from the iliac veins, though there was no focus of suppuration in the pelvis. There was some oedema of the feet and legs.

Escape of bile from the fistula of the operation wound is not very uncommon, but it is rare for all the bile to pass in this way, as in three cases mentioned by Godlee.

Lardaceous disease may supervene as the result of long-continued discharge of pus.

Diagnosis.—There is no one sign or symptom which may not be absent in tropical abscess. The most frequent indications are pain in the region of the liver, progressive enlargement of the organ, and fever; but these may be present in congestion, acute hepatitis, and other conditions. The presence of a fluctuating swelling or oedema of the abdominal wall over the liver makes the diagnosis almost certain, but unfortunately they are often, and in the case of oedema generally, absent. The history of dysentery or of residence in tropical countries, manifest illness, and the presence of some of the commoner signs and symptoms of abscess, such as hepatic pain, enlargement, and tenderness, and continued fever in the absence of any evidence of malaria or other sufficient cause, are strong grounds for suspecting a tropical abscess.

A considerable leucocytosis is of value in cases in which there is no other proof of abscess (Boinet,[7] Rogers[8]), but the leucocytosis may be comparatively slight or absent (Osler[9]). In a doubtful case leucocytosis is in favour of abscess, but its absence does not exclude abscess.

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[1] Novis. *Brit. Med. Journ.*, 1911, ii, 1408.
[2] Godlee. *Med.-Chir. Trans.*, 1902, lxxxv, 119.
[3] Rogers, L. *Brit. Med. Journ.*, 1903, i, 1316.
[4] Godlee. *Med.-Chir. Trans.*, 1902, lxxxv, 121.
[5] Pheasants. *Johns Hopkins Hosp. Bull.*, 1909, xx, 292.
[6] Rixford. *Occid. Med. Times*, 1895, ix, 415.
[7] Boinet. *Compt. rend. Soc. Biol.*, Paris, 1900, 11. s., ii, 1089.
[8] Rogers, L. *Brit. Med. Journ.*, 1905, ii, 1291.
[9] Osler. *Med. News*, N.Y., 1902, lxxx, 673.

Puncture of the liver with a trocar in order to determine if pus is present, is often employed to settle the diagnosis. It is not, however, in spite of statements to the contrary, free either from fallacy or from danger, especially from haemorrhage in malarial cases. The trocar may, of course, miss the abscess or may pass through it, or may get blocked and not bring any pus away. Death has followed exploratory puncture from haemorrhage (Hatch,[1] Maitland[2]); in order to diminish this risk, Maitland urges that when a fruitless puncture has been made, a second puncture in another direction should not be made while the end of the trocar is still in the liver, since this manoeuvre tends to enlarge the orifice and so favours haemorrhage; if necessary, an entirely fresh puncture should be made. Cantlie,[3] who strongly advocates the use of the aspirating syringe, considers that the inferior vena cava is the only vessel likely to be wounded, and that if the needle is not introduced for more than 3½ inches, this danger can be eliminated. The risk from haemorrhage is said to be greatest in cases in which there is no abscess. Further, a puncture may lead to peritonitis or may be the means of spreading infection in the liver by passing through the abscess and carrying the pus into other parts of the organ. When performed, aspiration should be employed as a means of localising the position of an abscess immediately before operation, and not merely for diagnosis.

Differential Diagnosis.—Suppurating Hydatid; Subdiaphragmatic Abscess; Pylephlebitis and Multiple Abscess; Suppurative Cholangitis; Intermittent Hepatic Fever; Pancreatic Cyst, Acute Pancreatitis; Suppuration in Rectus Muscle; Acute Hepatitis; Enteric Fever; Malaria; New Growth; Lymphadenoma; Acute Leukaemia; Gumma; Ascites; Dilated Gall-Bladder; Right Pleural Effusion and Empyema; Tuberculosis.

Suppurating Hydatid Cysts.—In simple hydatid disease the liver is enlarged but the general health and nutrition are good, there is no fever; and the general absence of symptoms, especially of pain, forms a striking contrast to the prominent local physical signs. No difficulty is therefore likely to arise in distinguishing between an ordinary hydatid cyst and abscess of the liver. When, however, a hydatid cyst suppurates, the condition of affairs is exactly the same as an abscess. In such cases the history that there had been a quiescent tumour in the position of the liver for a considerable time before the onset of symptoms would suggest the true state of affairs; otherwise it will be impossible to make an accurate diagnosis before operation. Difficulty might conceivably arise in diagnosing a simple hydatid complicated by fever from an abscess of the liver.

A *subphrenic abscess* may be due to various causes, such as leakage of an hepatic abscess or suppurating hydatid cyst, suppurative cholecystitis, perforation of a gastric or duodenal ulcer, appendicitis, calculous or tuberculous disease of the kidney, and in rare cases from suppuration in

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[1] Hatch. *Ind. Med. Gaz.*, 1898, xxxiii.

[2] Maitland. *Brit. Med. Journ.*, 1902, i, 458.

[3] Cantlie, J. *Ibid.*, 1903, ii, 656.

the spleen or in connexion with malignant disease of the large intestine. When an abscess, either tropical or one of the multiple abscesses seen in suppurative pylephlebitis or cholangitis, leaks on to the convexity of the liver and gives rise to a localised abscess between the diaphragm and the liver, the condition of affairs is for all practical purposes much the same as the original intrahepatic disease. When a localised peritoneal abscess is due to perforation of a gastric or duodenal ulcer, the cavity contains air and is often termed a subphrenic pyopneumothorax. The physical signs—resonance on percussion, a bell note, and succussion—distinguish it from hepatic abscess. A non-gaseous subphrenic abscess may be extremely difficult to differentiate from an abscess in the liver. Thus, in neglected appendicitis a large abscess may pass upwards from the right iliac fossa, displace the right lobe of the liver forwards, and cause increase in the hepatic dulness and apparent enlargement of the liver. It should be differentiated from an hepatic abscess by the history, the signs of appendicular mischief in the right iliac fossa, and by its tendency to bulge more into the right loin. The history or evidence of recent appendicitis is of some importance, for subphrenic abscess is commoner than hepatic abscess as a result of appendicitis (Elsberg [1]).

Suppurative Pylephlebitis and Multiple Abscesses.—The general resemblance of single and of multiple abscesses is often very close and an absolute diagnosis between them is not always justified. The points on which a distinction can be based are (i) local signs of abscess, such as fluctuation over a prominent swelling, or oedema of the body wall over the liver; (ii) the history of dysentery in the case of single abscess, or of appendicitis in multiple pylephlebitic abscesses. Bacillary dysentery, however, may be followed by multiple abscesses. In a patient who has had dysentery, agglutination of the Shiga-Flexner bacillus would point to bacillary dysentery and to multiple rather than single intrahepatic suppuration.

In a case, seen on March 22, 1903, at Coltishall, multiple abscesses, secondary to appendicitis, very closely resembled a single abscess. A man aged forty-eight was seized seven weeks before with pain at the umbilicus which lasted for one day; since that date there had been continued fever. There were dulness, friction, and distant bronchial breathing at the right base, but no enlargement of the liver in front. His temperature had fallen that morning to 96·4°, he was collapsed, looked almost moribund, and had a pulse of 140. A transpleural operation let out serous fluid from the pleura, and opened an abscess about the size of a teacup in the back of the right lobe of the liver; no other abscesses could be seen. The man seemed much better and the temperature remained below normal for four days; it then became hectic and he died on April 2. At the necropsy the vermiform appendix was gangrenous and the right lobe of the liver riddled with abscesses.

Intermittent Hepatic Fever.—In intermittent hepatic fever, depending on infective cholangitis due to calculi in the ducts, periodical attacks of fever, jaundice, pain, and hepatic enlargement occur. The history in such cases would probably point to cholelithiasis, and the variation in the size of the liver or absence of enlargement, the periodicity of the jaundice, and the intervals of fair health, would eliminate hepatic abscess. Infective cholangitis may, however, go on to suppuration and the temperature is then continuously raised.

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[1] Elsberg. *Ann. Surg.*, 1901, xxxiv, 729.

A *pancreatic cyst* lifting up the left, or less often the right, lobe of the liver may imitate an abscess in the substance of that viscus, but it is not necessarily accompanied by fever and often appears after a blow. But in very exceptional instances an abscess in the liver is not accompanied by pyrexia and may of course supervene after trauma. As a rule, a pancreatic cyst forms a definite tumour in the left hypochondrium of large size, and, as shewn by inflation of the stomach, lies behind that viscus.

Acute pancreatitis with the production of an abscess limited to the lesser sac of the peritoneum may, as shewn in the following case, resemble an abscess in the left lobe of the liver.

A man aged forty came into St. George's Hospital on September 15, 1899, having been ill sixteen days, with a fluctuating tumour in the epigastrium which had been noticed four days before. It was in the position of the left lobe, but the rest of the liver shewed no enlargement and the patient had never been out of England. A tentative diagnosis of suppurating hydatid cyst or of a pancreatic cyst was made. Mr. G. R. Turner operated the same day and opened a large abscess which was not in the liver but occupied the lesser sac of the peritoneum and went back to the spine. The patient did well for a time, but in October fever returned, he had rigors, and eventually died on November 8. The necropsy shewed an abscess cavity in the lesser sac of the peritoneum, gangrene of the tail of the pancreas, suppurative pylephlebitis, multiple abscesses in the liver, and a left-sided empyema.

Suppuration in the rectus muscle is rare, and when it does occur, as it has been known to do after typhoid fever, is usually below the umbilicus. An abscess in the rectus muscle above the umbilicus might imitate a pointing hepatic abscess, but there is not complete dulness over the swelling and the liver is not enlarged. A needle introduced into an abscess in the abdominal wall remains stationary, whereas a needle projecting into an abscess in the liver should move with respiration (Middeldorff's method). This test may fail when the liver is firmly adherent to the abdominal wall (Osler[1]), and is not devoid of danger, since leakage may take place from the puncture in the liver.

A *phantom tumour* is likely to be mistaken for an abscess only when the patient has been in the tropics or has been exposed to dysentery. The gradual disappearance of a phantom tumour under a general anaesthetic is important in the diagnosis from hepatic abscess. Bennett[2] has described, in a man who had had dysentery, a phantom tumour which was operated upon.

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[1] Osler. *Practice of Medicine*, p. 516, 6th ed., 1905.

[2] Bennett, W. H. *Lancet*, 1902, i, 3.

Since *acute hepatitis* precedes and may be considered an early stage of abscess, the question often arises whether suppuration has actually developed or not in a patient who has had dysentery and presents symptoms suggesting suppuration in the liver (*vide* p. 116).

In obscure forms of septicaemia the association of fever and enlargement of the liver from cloudy swelling may easily lead to a diagnosis of abscess. Remlinger[1] and Bozzolo described a form of acute hepatitis as the infectious liver which imitates abscess very closely.

In *enteric fever* considerable hepatic enlargement sometimes occurs and may give rise to a diagnosis of hepatic abscess.[2] This difficulty is more likely to arise in warm climates, where the two diseases are both prevalent. The presence or absence of the agglutination reaction should be of very great assistance, but it might be present in a patient with hepatic abscess who had previously had enteric fever. An examination of the blood should always be made, since there is no leucocytosis in uncomplicated enteric fever, whereas in abscess it may be present and be very considerable.

From *malaria* hepatic abscess may be diagnosed by examination of the blood for the parasite. In malarial cases treated with quinine the blood does not shew the parasite; in such cases leucopenia points to malaria, whereas in hepatic abscess there may be a high leucocytosis. Cases of hepatic abscess with little enlargement of the liver may easily be regarded as malarial in the absence of a blood examination; conversely, chronic malaria with hepatitis may imitate hepatic abscess, in the enlargement, pain both in the liver and the right shoulder, and the general ill-health. In malaria the hepatic enlargement is more uniform and is accompanied by enlargement of the spleen.

Influenza is sometimes accompanied or followed by hepatic enlargement and a continued temperature strongly suggesting suppuration; Goodhart[3] refers to such cases and I have had a similar experience.

New Growth.—Confusion sometimes arises when rapidly growing malignant disease in the liver is associated with a raised temperature. A soft growth, especially when in the left lobe, may cause a bulging of the abdominal wall and give rise to a sense of fluctuation more or less obscure. In such cases (*vide* p. 522) exploration is the only sure means of diagnosis. It is very rare indeed for the converse to occur, viz. for an abscess to be regarded as a new growth. It is more likely to occur in cases in which the patient is extremely cachectic and deeply jaundiced.

In a case of Osler's[4] the probable diagnosis was gall-stones with cancer. Woman, sixty-four years, dyspepsia for two years, loss of weight, pain in right side at intervals for three months, attacks of vomiting, slight jaundice, progressive weakness, no rigors, no leucocytosis, increase in size of liver, slight fever. Death. Amoebic abscess in the right lobe of liver. No ulceration of intestines.

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[1] Remlinger. *Presse méd.*, 1903, p. 86.

[2] Bozzolo, *Riv. crit. di clin. med.*, 1902, iii, 169; H. Jones, *Brit. Med. Journ.* 1897, ii, 1581.

[3] Goodhart. *System of Medicine* (Allbutt and Rolleston), 1905, i, 964.

[4] Osler. *Med. News*, N.Y. 1902, lxxx, 673.

In generalised *lymphadenoma* the temperature may closely resemble that of suppuration, and in rare instances there is very considerable enlargement of the liver.

Some years ago a man was under my care with a pedunculated mass in the left groin of many years' standing, which turned out to be lymphadenoma; he developed a hectic temperature and considerable enlargement of the liver. The condition was not unlike hepatic abscess and the question of operation was raised during life. At the necropsy the liver shewed lymphadenoma. I have seen a case in which lymphadenoma of the liver and mesenteric glands with very slight enlargement of the superficial lymphatic glands had been operated upon for hepatic abscess.

As a rule, there is not much enlargement of the liver in generalised lymphadenoma. In lymphadenoma there is usually no leucocytosis, whereas in abscess there may be a polymorphonuclear leucocytosis. Further, in lymphadenoma the superficial glands are usually affected.

Acute leukaemia, especially in the absence of glandular enlargement, may imitate hepatic abscess unless a blood examination is available (Emerson,[1] Kelly [2]).

In some cases of *syphilis* there is continued fever (*vide* p. 364) and very considerable hepatic enlargement; in patients who have been in countries where hepatic abscess is common these manifestations may easily lead to an erroneous diagnosis.[3] Cases of hepatic gumma have been operated upon for abscess.

Ascites.—In a few cases an abscess has been so large as to imitate ascites, and the abdomen has been tapped on this assumption. This is more likely to occur in young subjects. Cases have been recorded by Hatch [4] and Powell.[5]

Though it seems unlikely, a greatly *dilated gall-bladder* has been tapped under the idea that it was an hepatic abscess (*vide* specimen (No. 1381) in St. Thomas's Hospital Museum).

Right-sided Pleural Effusion.—There may be a very close resemblance between hepatic abscess and a right-sided pleural effusion. An abscess in the upper and back part of the right lobe of the liver will push the diaphragm up and give rise to dulness at the base of the lung. The dulness is said to be curved and to be higher in front and in the axilla than behind, but this is not constant, as there may be a small concomitant pleural effusion in the right pleura due to the spread of inflammation through the diaphragm. In a right-sided pleural effusion Grocco's paravertebral triangle of dulness is present on the left side; in 32 cases

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[1] Emerson. *Johns Hopkins Hosp. Bull.*, 1907, xviii, 80.

[2] Kelly, A. O. J. *Trans. Assoc. Amer. Phys.*, 1903, xviii, 481.

[3] Compare Barry, C. *Ind. Med. Gaz.*, 1904, xxxix, 298.

[4] Hatch. *Ibid.*, 1898, xxxiii, 285.

[5] Powell. *Ibid.*, 1898, xxxiii, 41.

of hepatic abscess Castellani[1] found that Grocco's triangle was absent. The upward displacement of the diaphragm does not displace the heart to the left in the same way that a large pleural effusion does, but this is not of much use, as hepatic abscess rarely imitates a large pleural effusion. In hepatic abscess the patient's general condition is worse than it would be in a comparatively small empyema or pleural effusion; further, the liver is usually enlarged downwards in abscess, whereas a pleural effusion of the size which hepatic abscess imitates would not displace the liver downwards. As has been pointed out, hepatic abscess often gives rise to pleurisy and pleuritic pain, so that the question to be decided is whether there is an abscess in the liver or whether the whole disease is intrapleural. In a case of tropical abscess following enteric fever (*vide* p. 122) the resemblance to an empyema was considerable.

Cases of *pulmonary tuberculosis* with hectic temperature and with an enlarged fatty liver, in which the physical signs of pulmonary disease are not prominent or have not been detected, have been diagnosed as hepatic abscess.[2] In hepatic abscess without any hepatic enlargement in patients who have never been out of England the continued temperature may, in the absence of any other signs, suggest generalised tuberculosis.

The **prognosis** of hepatic abscess, though always serious, and specially severe in the presence of complications, which will be referred to later, is by no means necessarily bad.

In a collection of 1094 cases the mortality was 30 per cent (Solonoff[3]).

Lafleur[4] considers the prognosis more unfavourable in amoebic than in other forms of hepatic abscess, but Manson[5] joins issue on this point. The prognosis is considerably modified by the period at which operative interference takes place. In cases operated upon early, the outlook is favourable. This probably explains why operation in private practice in India is much more successful than in hospital.

According to Moorhead[6] 75 per cent of private cases and 50 per cent of hospital cases recover. Hatch[7] found that in some years 90 per cent of the hospital cases terminated fatally, while in private practice the mortality was about 20 per cent.

When operation is undertaken late, the prognosis is bad, inasmuch as extensive destruction of the liver substance has taken place, the patient's strength is exhausted by the continued fever, and secondary results, such as pyaemia and emaciation, have had time to develop.

Rupture of the abscess externally is favourable; rupture into the lung is generally regarded as fairly favourable, though convalescence may be tedious; thus, De Castro[1] estimates that 76 per cent recover, and Manson[2] that spontaneous recovery occurs in 50 per cent; but Lafleur and Godlee take an unfavourable view of this complication. Rupture into the colon is usually followed by recovery. On the other hand, rupture into the general peritoneal cavity and into the pericardium are nearly always fatal. The presence of active dysentery or diarrhoea is unfavourable. The existence of more than one abscess makes the prognosis bad; this is difficult to diagnose, but it should be suspected if the temperature remains raised when an abscess has been opened and is discharging quite freely. When the abscess continues to discharge for a long time and signs of lardaceous disease—albuminuria, oedema of the feet, and diarrhoea—appear, the outlook becomes grave.

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[1] Castellani. *Riv. crit. di clin. med.*, Firenze, 1908, ix, 375.
[2] Josserand. *Lyon méd.*, 1897, lxxxvi, 421.
[3] Solonoff. *Brit. Med. Journ.*, 1903, i, 262.
[4] Lafleur. Allbutt's *System of Med.*, 1897, iv, 168.
[5] Manson, P. *Tropical Diseases*, p. 457, 1903.
[6] Moorhead. *Brit. Med. Journ.*, 1899, i, 1032.
[7] Hatch. *Lancet*, 1902, ii, 1543.

After apparent recovery from hepatic abscess there is a danger of recurrence, more especially if the patient remains in a tropical country or returns to it too soon after recovery.

Treatment.—*Prophylaxis* of hepatic abscess is in the first instance the treatment of acute hepatitis (*vide* p. 116). Alcohol should be tabooed and care taken by the subjects of past dysentery to avoid chills and recurrences of diarrhoea. ^ ~~Large doses, 20-40 grains daily, of ipecacuanha are~~ invaluable in amoebic hepatitis as a means of preventing suppuration ^ ~~(Rogers,[3] Pilgrim,[4] Greig and Wells[5]). The dose should be taken at night on an empty stomach; to prevent vomiting chloral or chloretone should be given before the ipecacuanha, or the latter may be taken in keratin-coated pill~~s. The patient should be kept in bed on a low diet. Dysentery should of course be avoided by attention to the drinking-water. The bowels should be kept open by mild laxatives, and local pain relieved by leeches, cupping, the application of an ice-bag, and other methods mentioned in the treatment of acute hepatitis. Chloride of ammonium in xx.-grain doses three times a day has been recommended, but its value is somewhat problematical. ^ ~~Removal~~ of blood from the congested organ ~~by~~ aspiration has been thought to ~~be of use in~~ prevent~~ing~~ suppuration, but, as already pointed out, it is not entirely free from risk (*vide* p. 146).

The *treatment* of hepatic abscess is essentially surgical and consists in removal of the pus and free drainage of the abscess cavity at the earliest opportunity. Medical treatment is only justifiable when it is doubtful if an abscess has formed, and during the period when acute hepatitis will explain the symptoms; and, apart from symptomatic remedies, has been dealt with under "prophylaxis."

Aspiration.—Tapping the liver through the abdominal walls with a Dieulafoy's aspirator has been commonly advocated and is often successful. It is, however, dangerous, and should not be attempted. The

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[1] De Castro. *Les Maladies du foie dans les pays chauds*, Paris, 1870.

[2] Manson. *The Lane Lectures on Tropical Diseases*, p. 192, 1905.

[3] Rogers. *Med.-Chir. Trans.*, Lond., 1907, xc, 145.

[4] Pilgrim. *Ind. Med. Gaz.*, 1910, xlv, 332; *Brit. Med. Journ.*, 1910, ii, 2039.

[5] Greig and Wells. *Scientific Memoirs by Officers of the Medical and Sanitary Departments of the Government of India*, 1911, N.S., No. 47.

∧/ Emetine 2/3 grain, hypodermically, twice daily or better emetine bismuth iodide grs III in salol coat pills for 12 nights

∧/, and should be tried in every case of suspected abscess

Rogers finds that injection of $\frac{1}{2}$ gr of emetine hydrochloride into the abscess cavity gives better results.

Among 49 cases treated by aspiration and hypodermic injection of a large dose of emetine Thurston obtained 40 cures

Rogers, L. *Brit. med. Journ.*, 1912, ii, 406.

Thurston, E.O. *Lancet*, 1924, ii, 1008.

abscess may leak into the general peritoneal cavity and set up general peritonitis; severe haemorrhage may occur if the aspirator wounds a large branch of the hepatic artery, or possibly the aspirator may perforate one of the hollow abdominal viscera, such as the stomach or intestines.

In England Manson and Cantlie[1] have enthusiastically advocated drainage of the abscess by means of a trocar and cannula, and establishing a syphon drainage. This method has not found favour with Indian surgeons. It is urged that this method does not provide thorough drainage, that if the tube becomes displaced there is great difficulty in replacing it, and that if the tube gets out the pus may escape into the peritoneal cavity.

In amoebic abscesses containing no pyogenetic organisms cure has followed aspiration and injection of a solution containing 30 grains of the acid bihydrochloride of quinine into the abscess cavity (Rogers and Wilson[2]).

The most satisfactory treatment of an abscess is free opening and drainage. For the surgical procedure the reader should refer to surgical textbooks. The position of the abscess must be first determined, and though it is not a harmless procedure, this must often be done by exploration with an aspirator under an anaesthetic. The abscess should be freely opened so as to allow of its interior being explored and any adjacent abscess opened. It is essential that adequate provision for free drainage should be made.

When an abscess bursts into the pleura or peritoneum operation should be undertaken, and in the case of the peritoneum of course without delay (*vide* p. 143). The treatment of rupture into the lung requires consideration; some authorities urge immediate operation in order to prevent further destruction of the lung. Others advise that the case should be treated with vaccins and an open-air life, and that if improvement is steady no surgical steps are needed, but if the symptoms persist operation should be advised. Drainage of an abscess through the lung has been satisfactorily treated by placing the patient in the upside-down position so as to facilitate drainage (M'Kechnie[3]). In rupture into the colon no operation is necessary unless there are signs of peritonitis. Operation should be undertaken in the rare cases in which rupture occurs into the pericardium or pelvis of the kidney, but in the former event death may be very rapid.

During *convalescence* tonics, fresh air, and nourishing food are necessary. Change of air to the seaside should be recommended. The patient should not remain in a tropical climate, or return until two years have elapsed since the abscess was opened, and if possible should remain permanently in England or a temperate climate.

[1] Cantlie, J. *Brit. Med. Journ.*, 1903, ii, 656.
[2] Rogers and Wilson. *Ibid.*, 1906, i, 1397.
[3] M'Kechnie. *Lancet*, Lond., 1912, i, 865.

MULTIPLE ABSCESSES IN THE LIVER

THE infection leading to multiple abscesses in the liver may reach the organ in several ways. The method of production provides a means of classifying multiple hepatic abscess.

(1) Ordinary pyaemic abscesses, in which the infection arrives by the hepatic artery.

(2) Abscess due to portal vein infection. (*a*) Multiple abscesses in bacillary and amoebic dysentery, in appendicitis, etc., due to infective emboli. (*b*) Suppurative pylephlebitis (*vide* p. 68).

(3) Suppuration in and spreading from the bile-ducts. This is described under "Suppurative Cholangitis," and includes verminous abscesses due to worms which have travelled up the common bile-duct.

(4) Secondary abscesses due to the spread of infection from a large single abscess.

Pyaemic Abscesses.—In general pyaemia numerous minute abscesses may be found in the liver.

In 24 cases of general pyaemia that were examined in the post-mortem room of St. George's Hospital, 1890–1896, abscesses were found in the liver in 4 cases, multiple in 3, in the remaining case there were two abscesses.

In infective endocarditis secondary abscesses in the liver are rare; thus, in 65 fatal cases of infective endocarditis collected by Kelynack[1] there was only one case in which the liver was affected. In 10 cases of systemic blastomycosis examined after death the liver shewed small suppurating foci in 4 (Montgomery and Ormsby[2]).

An hepatic abscess may be secondary to suppuration anywhere in the body, even when there is no pyaemia. In such cases the liver is probably in a state of diminished resistance, so that micro-organisms which would be destroyed elsewhere are able to flourish there. Percival Pott long ago stated that multiple abscesses in the liver were especially apt to follow injuries and suppuration in the head.

The commonest causes of pyaemia, which is a comparatively infrequent disease, are acute infective periostitis and middle-ear disease. In cases of middle-ear disease multiple hepatic abscesses may be merely pyaemic. But it is noticeable that sometimes abscesses are not found in any other organ. This is difficult to explain. It might be said that the micro-organisms manage to get through the lungs without being arrested there and that the liver is the place of least resistance, the other organs in the body destroying the micro-organisms. Another conceivable explanation is that the micro-organisms pass down from the head and drop,

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[1] Kelynack. *Encyclopaedia Medica*, 1900, iv, 365.

[2] Montgomery and Ormsby. *Arch. Int. Med.*, Chicago, 1909, ii, 12.

so to speak, through the right auricle into the hepatic veins. This passage of micro-organisms against the current of blood or retrograde embolism is rare, but it undoubtedly occurs.[1] It is possible that in the pre-anaesthetic days of Pott, retrograde infection of the liver from suppuration about the head was commoner than at the present time, when operations are quietly performed under an anaesthetic. Thus retrograde embolism is favoured by violent expiratory efforts which may result in blood, and the pus or micro-organisms in it, being driven out of the right auricle into the hepatic veins. Such expiratory efforts would be likely to occur at the time of any operation in the pre-anaesthetic days.

Multiple Abscesses due to Portal Vein Infection.—Multiple abscesses may be due to widespread embolism of the intrahepatic branches of the portal vein. When this occurs without any pylephlebitis of the extrahepatic parts of the portal vein, the process in the liver may be regarded as due to "portal pyaemia." In reality no hard-and-fast line can be drawn between such cases and ordinary pylephlebitis with multiple foci of suppuration in the liver.

Multiple abscess may occur ~~in dysentery. It is rare~~ in amoebic ~~rather less so~~ in bacillary dysentery. In both these forms of dysentery there may be a number of small abscesses all of about the same size and due to multiple embolism of the intrahepatic branches of the portal vein. There may be a single large abscess which has, by infection of the branches of the portal vein, given rise to secondary and multiple foci of suppuration.

and

In amoebic dysentery the contents of the small multiple abscesses are barely liquid. The walls are formed of necrotic liver tissue and ~~the abscesses have evidently originated in a portal space.~~ There is ~~usually~~ absence of the ordinary small-celled infiltration seen in other forms of suppuration ~~(Lafleur[2]). According to Rogers,[3] the small multiple abscesses are due to~~ a mixed infection of amoebae and pyogenetic cocci, while the large abscesses are solely due to amoebae. ~~Rogers figures small-celled infiltration in the small multiple abscesses. Multiple abscesses containing amoebae have been found in cases in which the intestines appeared normal (Buxton[4]).~~

except wh
there is

In bacillary dysentery the multiple abscesses are like those in pylephlebitis. In the dysentery seen in South Africa in the war of 1899–1902 hepatic suppuration, when it occurred, was usually in the form of multiple liver abscesses without pylephlebitis.

Multiple abscesses ~~from portal vein infection are most commonly~~ secondary to appendicitis. ~~The abscesses~~ may be due to multiple infective emboli which have passed up from a small abscess in connexion with the appendix, the intervening part of the portal and superior mesenteric veins being normal; there may be as many as 200 abscesses.

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[1] For examples *vide* Welch, Allbutt's *System of Medicine*, 1899, vi, 233; Risel, *Virchows Arch.*, 1905, clxxxii, 258.

[2] Lafleur. Allbutt's *System*, 1897, iv, 158.

~~[3] Rogers, L. *Brit. Med. Journ.*, 1902, i, 1317.~~

~~[4] Buxton. *Proc. Phila. Path. Soc.*, 1899, ii, 49.~~

In some instances there is a large abscess with smaller ones around it; the large abscess may be due to originally separate abscesses running together. Leakage or rupture of a small abscess causes general or localised peritonitis; in the latter event there may be an abscess between the convex surface of the liver and the diaphragm. Either an empyema on the right side or merely pleurisy with effusion is a frequent complication. Luckily hepatic suppuration is not a common complication of appendicitis; it is more likely to occur with a small abscess under considerable tension (*vide* also p. 69).

In 112 cases of appendicitis examined after death there were 2 cases of abscess of liver, 2 cases of suppurative hepatitis, and 2 of perihepatitis (Langheld[1]).

The following is a good example of Dieulafoy's "appendicular liver":—

A woman aged twenty-four with a history of appendicitis presented signs of a local abscess in the right iliac fossa; the appendix, which was thickened, and an ounce of pus in its neighbourhood were removed. The patient went on well for a week, when the discharge from the wound became very profuse; exploration shewed that there was a suppurating cavity lined by coils of intestines. Her condition became one of chronic pyaemia, and in the last seven weeks of life the temperature was only normal on four occasions. At the necropsy the right lobe was riddled with abscesses; there was no general pylephlebitis or thrombosis of the portal vein, but a few intrahepatic branches of the portal vein in the right lobe were thrombosed.

Sometimes the existence of the primary source of infection in the appendix is entirely latent and the patient comes under observation with a hectic temperature, rigors, and an enlarged liver.

Multiple abscesses of the liver are very seldom secondary to gastric ulcer; Murchison[2] recorded two cases. Suppurative pylephlebitis is also extremely rare after gastric ulcer. In Lambert's[3] curious case a pin passed from the stomach into the right lobe of the liver and set up multiple abscesses. In very rare instances, of which Gibbon[4] has collected nine examples, multiple abscesses occur in the liver after typhoid fever; they may be secondary to intercurrent appendicitis and pylephlebitis (Osler[5]) due to embolism of the small branches of the portal vein, or to infection through the hepatic artery and secondary to an abscess elsewhere in the body.

In a case reported by Mackie Whyte[6] multiple hepatic abscesses without any pylephlebitis were due to infection derived from a false diverticulum of the sigmoid flexure. In a case of primary ulceration of the lower part of the

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[1] Langheld. Quoted by Loison, *Rév. de chir.*, Paris, 1900, xxi, 522.

[2] Murchison. *Trans. Path. Soc.*, 1866, xvii, 145.

[3] Lambert. *New York Med. Journ.*, 1898, lxvii, 177.

[4] Gibbon, J. H. *Am. Journ. Med. Sc.*, 1903, cxxv, 592.

[5] Osler. *Trans. Assoc. Am. Physicians*, 1897, xii, 382.

[6] Mackie Whyte. *Scot. Med. and Surg. Journ.*, 1906, xviii, 120.

Suppuration has been reported in connection with a fish bone (Crossan), a straw (Belfrage), a piece of glass (Barling)

Crossan. Med. and Surg. Rep. Episcop. Hosp. Phila., 1916, IV, 304
Belfrage. Upsala LÄKAREförening Förhlan~~ga~~dlingar 1874-5, X, 150.
Barling. Arch. Roentg. Ray 1915, XIX, 25.

Multiple abscesses in the liver, containing *Oidium roseum non-liquefaciens* and bacteria like *B. coli* were regarded as secondary to gastro-enteritis (ZENONI).

L. Rogers has reported the cure by emetine of multiple amoebic abscesses seen at laparotomy

ZENONI. *Sperimentale*, FIRENZE, 1912, LXVI, 33

Rogers, L. *Trans. Med. Soc. London*, 1924, XL, 13 rectum multiple abscesses in the liver, containing *Bacillus influenzae similis*, were found by Ophüls.[1] There was no thrombosis in the haemorrhoidal or portal veins.

Multiple abscesses in the liver may be due to suppuration in the pelvis, the portal vein being infected through the communications between the superior haemorrhoidal branch of the inferior mesenteric vein and the branches (middle and inferior haemorrhoidal) of the internal iliac veins.

Thus, a prostatic abscess (Lancereaux[2]), gonorrhoeal salpingitis (Handford[3]), perimetric abscess (Roughton[4]), and a suppurating submucous fibromyoma (Delestre[5]) have been known to cause multiple abscesses in the liver.

Secondary Abscesses due to the Spread of Infection from a Large Single Abscess.—Small abscesses are often found associated with a large single abscess cavity. In some instances the large abscess is due to confluence of small abscesses; in other instances the small abscesses are secondary to infection from the originally single abscess, and are usually found in its neighbourhood. They were described by Davidson[6] under the name of "secondary pyo-septicaemic abscess."

The clinical features, diagnosis, and prognosis in cases with the larger multiple abscesses in the liver are practically the same as in suppurative pylephlebitis, to which the reader should refer. The miliary abscesses in the liver in general pyaemia do not give rise to any special symptoms.

PERIHEPATITIS

Perihepatitis, or inflammation of the peritoneal capsule of the liver, may be acute or chronic.

Acute Perihepatitis

Causation.—In temperate climates acute inflammation of the peritoneum covering the liver and the underlying capsule is not a primary and independent condition, in the same way that pericarditis and pleurisy often are, but is secondary to disease in the liver or in the neighbourhood, and is usually quite subordinate to the primary affection. The only exception to this is the occurrence of traumatic perihepatitis such as follows fracture of the ribs on the right side; in such cases acute inflammation of the peritoneum covering the bruised or wounded areas of the liver may be found if there is a fatal termination. It has been thought (Cantlie[1]) that in the tropics it may be a primary affection like acute pleurisy; but it is reasonable to believe that its frequency in hot climates depends on the fact that active congestion of the liver and acute hepatitis, which are common, often extend to the surface of the liver and thus set up perihepatitis.

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[1] Ophüls. *Am. Journ. Med. Sc.*, 1901, cxxii, 797.

[2] Lancereaux. *Traité des maladies du foie et du pancréas*, p. 231, 1899.

[3] Handford. *Trans. Path. Soc.*, 1886, xxxvii, 267.

[4] Roughton. *St. Barth. Hosp. Rep.*, 1885, xxi, 173.

[5] Delestre. *Bull. Soc. anat.*, Paris, 1898, lxxiii, 219.

[6] Davidson. Allbutt's *System of Med.*, 1897, iv, 133.

Acute perihepatitis may be secondary to acute hepatitis, gummas, suppuration inside the liver, such as abscess, suppurating hydatid cyst, pylephlebitis, and cholangitis. It may also be seen over nodules of new growth involving the surface of the liver.

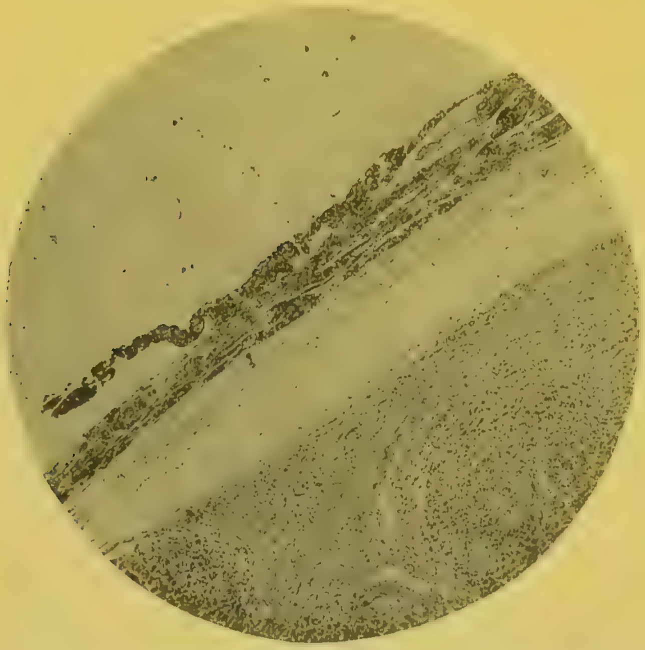
FIG. 22.—Lymph in acute perihepatitis on the surface of a cirrhotic liver which shews some thickening of the capsule. The lymph appears opaque. Some separation has taken place between the layers of the thickened capsule. (Photomicrograph by Dr. S. G. Penny.)

Acute inflammation of the peritoneal surface of the liver necessarily occurs in general peritonitis and in some forms of localised peritonitis due to inflammation, ulceration, and perforation of adjacent viscera; for example, in pancreatitis and inflammation localised to the lesser sac of the peritoneum, in cholecystitis, and in subphrenic abscess. In such conditions in which the surface of the liver happens to form the wall of an abscess cavity there is really no need to speak of pyoperihepatitis or pyopneumo-perihepatitis.[2] The first symptoms of a subphrenic abscess due to appendicitis may be those of acute perihepatitis.

In acute pleurisy, pneumonia, and pericarditis, inflammation may spread through the diaphragm and set up local peritonitis over the convexity of the liver. Occasionally it develops in the course of chronic venous engorgement of the liver, either as part of a passing, or of a terminal, infection of the liver. Acute perihepatitis is more often partial than universal.

Morbid Anatomy.—The surface of the liver in the affected area shews the dulling, loss of gloss, and slight granular appearance seen in acute peritonitis elsewhere. The vessels of the capsule are injected with blood

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[1] Cantlie, J. *Encyclopaedia Medica*, 1901, vii, 2.

[2] *Vide* Chauffard, in *Traité de médecine* (Bouchard-Brissaud), 1902, v, 154.

J. incequotitis

Tenderness on pressure between the two heads of the sterno-mastoid muscle on the right side has been described (CADE and PARTURIER)

Cade et Parturier *Rev. de méd.*, Par., 1919, XXXVI, 4 and the cells of the liver immediately under the capsule may shew cloudy swelling. At a later date organisation of the fibrinous lymph takes place and filamentous adhesions unite the liver to the diaphragm, abdominal wall, and the adjacent viscera. These adhesions are not progressive and must not be considered as evidence of chronic perihepatitis. Such adhesions are very common in the post-mortem room, and in the great majority of instances have not given rise to any symptoms during life. Exceptionally they induce pain and a sense of dragging in the right hypochondrium. If the adhesions involve the stomach, its movements may be so interfered with that dyspepsia of a very obstinate nature adhesion dyspepsia) may be produced.

The late Mr. Allingham performed laparotomy on a patient with very severe dyspepsia, under the care of Dr. Vernon Jones, and found firm adhesions between the stomach and liver; these were divided and the symptoms were relieved. There was a history of acute perihepatitis twelve years before in India.

As an example of perihepatitis due in all probability to a terminal infection the following case is of interest:—

A boy aged ten years died in St. George's Hospital with adherent pericardium, a dilated and hypertrophied left ventricle, and signs of backward pressure. In the last few days of life he had pain over the liver and a raised temperature, but no jaundice. At the necropsy there was recent lymph on the surface of the liver, which weighed 29 ounces. On section the liver was swollen, of a mottled yellow colour, not nutmeggy.

Signs and Symptoms.—There is pain over the liver, which is made worse on respiration and is accompanied by a friction rub. The right side of the chest moves little and the liver is tender to the touch, and when an attempt to examine it is made, the overlying abdominal muscles at once become rigid. If the liver is grasped between the two hands, placed in front and behind, and moved backwards and forwards, acute pain is elicited, which may run up to an area between the clavicle and the acromion process of the scapula on the front of the chest (Cantlie), thus differing from the shoulder pain of intrahepatic disease. ^ Dry cough may occur and be readily induced by examination of the liver. Fever, sense of weight in the hypochondrium, and other symptoms may be due to accompanying and underlying hepatitis and active congestion. Hiccup is not uncommon, and nausea and vomiting may occur. Occasionally there is ascites, but very rarely a sufficient amount to be detected clinically (compare p. 245).

Diagnosis.—Perihepatitis is practically always due to some underlying condition, and search must always be made for the primary disease, such as abscess and other forms of intrahepatic suppuration, hepatitis, new growth, chronic venous engorgement. Acute perihepatitis is very likely to be confused with acute pleurisy on the right side; indeed, in many cases the two conditions coexist, the inflammation spreading from one serous surface to the other. In differentiating these two conditions Cantlie lays stress on grasping the liver between the hands and moving it; pain running up into the supraclavicular fossa is regarded as diagnostic of perihepatitis.

Treatment.—Rest and warmth in bed are important, while the underlying cause is sought for and treated. Treatment of the inflamed capsule of the liver will consist chiefly in the relief of pain. If severe, six or more leeches may be applied and be followed by a poultice; in slighter cases hot fomentations over the liver, or counter-irritation in the form of a mustard poultice or leaf, will be sufficient. Dry cupping often gives considerable relief. Strapping the side, as in the method employed for fractured ribs, will minimise movement and pain. A light milk diet should be given at first until the patient feels able to take solid food.

Chronic Perihepatitis

Under this heading two conditions of very different importance are included—(i) local and (ii) universal perihepatitis.

Local Chronic Perihepatitis

This condition may be due to various causes. In its slighter degrees it is seen as the result of pressure in tight-laced livers, when a belt has been worn, or may result from the communicated pulsations of a large heart. The thickening of the capsule, often associated with some atrophy of the liver substance immediately subjacent, is analogous to the milk spots so commonly seen on the surface of the heart. In many cases this local thickening of the capsule is associated with backward pressure due to obstructive heart or lung disease; in such cases the distension, and perhaps pulsation, of the liver, by giving rise to increased friction and attrition, help to call forth the capsulitis. In 18 cases of Hale White's[1] backward pressure was present in 10. In 87 cases of tricuspid stenosis recorded by Pitt[2] perihepatitis was present in 11. The local perihepatitis is occasionally part of malignant or tuberculous peritonitis, or may be found over a gumma or hydatid cyst embedded in the liver; it may also be due to the irritation of a calculous gall-bladder or of a gastric ulcer. Capsulitis of the spleen is frequently associated with it (in 9 out of 18 of Hale White's cases).

Local perihepatitis very seldom goes on, so far as is known, to the diffuse or universal form. Nicholls[3] records a case of local perihepatitis due to cholecystitis in which this transformation was in progress. The thickened portion of the capsule of the liver does not peel off, but is firmly adherent; it thus contrasts with the "false membranes" seen in universal perihepatitis, which lie on the surface of, and not in the sub-

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[1] Hale White. *Trans. Clin. Soc.*, Lond., 1888, xxi, 219.

[2] Pitt. Allbutt's *System*, 1899, vi, 23.

[3] Nicholls, A. G. *Studies from the Royal Victoria Hosp.*, *Montreal*, 1902, i, 41.

stance of, the capsule of the liver. The local thickenings on the liver resemble anatomically the milk spots on the heart and the corneal fibromas on the capsule of the spleen. Microscopically they shew parallel strands of hyaline fibrous tissue with cells between them, but do not contain blood-vessels. These local areas of chronic capsulitis may be adherent to adjacent parts, but usually they are free.

Local chronic perihepatitis may account for some pain and tenderness over the liver and may possibly give rise to a friction rub over the organ. Strictly localised perihepatitis does not give rise to ascites; but in some cases in which areas of chronic perihepatitis are scattered over the surface of the liver so as to produce a transition between local and universal chronic perihepatitis, ascites may possibly be due to this cause. The cases of cirrhosis of the liver with ascites requiring frequent tappings may be due to this form of chronic peritonitis. Strictly localised chronic perihepatitis is of little or no clinical importance apart from the associated morbid conditions of the liver.

Treatment.—If the condition is suspected and there is pain, it may be treated by local applications, such as poultices, hot compresses, or belladonna plasters, but usually no special treatment is required.

UNIVERSAL CHRONIC PERIHEPATITIS

Synonyms: Diffuse Chronic Hyperplastic Perihepatitis, Chronic Hyaline Perihepatitis, Chronic Deforming Perihepatitis, Sugar-iced Liver (*Zuckergussleber*).

History, etc.—The condition was observed by Budd[1] in 1852. Curschmann[2] in 1884 described the morbid changes in detail and invented the graphic name of sugar-iced liver (*Zuckergussleber*). The writings of Hilton Fagge[3] and Hale White[4] have been of great value in distinguishing the clinical aspects of perihepatitis from those of cirrhosis, and in drawing attention to the relationship between chronic interstitial nephritis and chronic perihepatitis. Nicholls[5] (1902) published a monograph of 80 pages, and Kelly[6] wrote an exhaustive paper on this subject.

This condition has received a number of rather cumbrous names, but is usually only a local manifestation of diffuse chronic peritonitis. It is in fact artificial to separate chronic universal perihepatitis from diffuse chronic peritonitis. The chronic inflammatory change may begin in the peritoneal coat of the liver and subsequently involve the adjacent peritoneum, or it may extend to the serous covering of the organ in cases of more or less general chronic peritonitis. The name chronic perihepatitis thus suggests a more intimate relationship to the liver than actually exists. A more accurate though longer descriptive title would be chronic peritonitis with perihepatitis, or chronic peritonitis involving the liver.

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[1] Budd. *Diseases of the Liver*, p. 139.

[2] Curschmann. *Deutsche med. Wchnschr.*, 1884, x, 564.

[3] Hilton Fagge. *Principles and Practice of Med.*, 1886, ii, 294.

[4] Hale White. *Trans. Clin. Soc.*, 1888, xxi, 219; *Guy's Hosp. Rep.*, 1893, xlix, 1.

[5] Nicholls, A. G. *Studies from the Royal Victoria Hospital, Montreal*, i, No. 3, 1902.

[6] Kelly, A. O. J. *Amer. Jour. Med. Sc.*, 1903, cxxv, 116.

Pathogeny.—The chronic peritonitis, of which chronic perihepatitis is part, is usually that form spoken of as simple, since it is not manifestly due to tuberculosis or to new-growth. It is characterised by a widespread fibrosis with cicatricial contraction, so that the mesentery and omenta are shortened and the viscera become inclosed in a firm casing which contracts upon and compresses them. In considering the question of causation, it will be convenient to divide cases of chronic perihepatitis and peritonitis into three groups:—

(I) When associated with varying degrees of chronic inflammation of the pericardium and pleurae (multiple serositis).

(II) When associated with arteriosclerosis and granular kidneys.

(III) When associated with other conditions.

(I) *When associated with Varying Degrees of Chronic Inflammation of the Pericardium and Pleurae* (*Multiple Serositis*).—Chronic peritonitis and chronic universal perihepatitis may be associated with varying degrees of the same change in, or in connexion with, the serous membranes in the thorax—the pericardium and pleurae. The combined changes in more than one serous membrane are spoken of as multiple progressive hyaloserositis, polyorrhymenitis, or Concato's disease. There may be a descending scale in the extent of the associated intrathoracic changes. Thus, there may be extensive fibrosis of the mediastina with adherent pericardium and pleurae (indurative mediastino-pericarditis), adherent pericardium with thickening and obliteration, partial or complete, of both or of only one pleura, or only a calcified and adherent or an adherent pericardium. In this class the inflammatory change is most intense on each side of the diaphragm, and fades off in the more distant parts of the peritoneum and pleurae; probably the constant movement of the diaphragm assists in keeping up the morbid process when once it has been started. The inflammatory change beginning on one side of the diaphragm readily spreads through the lymphatics to the neighbouring serous membrane. In most cases the inflammatory change is primary in the pericardium or pleura and spreads to the convexity of the liver, but in some it is probable that it begins as an acute inflammation close to the convexity of the liver and subsequently becomes chronic and extends to the right pleura and pericardium. In chronic perihepatitis associated with intrathoracic changes of the same kind the kidneys are, as a rule, healthy, or merely shew chronic venous engorgement. But in some instances they are granular and thus merge into the next group.

A well-marked example of chronic proliferative peritonitis and perihepatitis associated with chronic indurative mediastino-pericarditis was seen in a man aged fifty-five years, in St. George's Hospital, in 1896. He presented ascites, which required tapping 26 times, and oedema of the legs; during life mediastinal growth appeared probable from the existence of post-sternal dulness.

At the necropsy the pericardium was firmly adherent to the heart and to all the surrounding parts, especially in the region of the thymus, which was occupied by very dense firm fibrous tissue (microscopically only fibrosis). There were very dense pleural adhesions, ½ an inch thick on the left side, and universal chronic peritonitis and perihepatitis. The liver, 72 ounces, microscopically shewed early cirrhosis, though to the naked eye it appeared normal. The kidneys, 8 ounces each, were healthy.

A number of cases of universal chronic perihepatitis associated with adherent pericardium have been recorded and the condition is not in reality rare.

Gilbert and Garnier[1] described 11 cases under the name of *symphyse péricardio-périhépatique.* Heidemann,[2] in a paper on the results of adherent pericardium, collected 7 fresh cases. Kelly referred to 27 cases of universal perihepatitis associated with adherent pericardium. I have seen 2 cases in which chronic perihepatitis was associated with, and probably due to, extension of inflammation through the diaphragm, from an adherent pericardium that had undergone calcification.

A condition closely allied to universal chronic perihepatitis associated with adherent pericardium was described by Pick[3] as pericarditic pseudo-cirrhosis of the liver, in which together with latent adherent pericardium there was ascites due to fibrous hyperplasia and circulatory disturbance in the liver. Any evidences of chronic inflammation of the peritoneum were regarded by Pick as accidental or secondary to ascites and chronic venous engorgement. Cases of this kind without chronic universal perihepatitis or chronic peritonitis certainly occur, and have already been described as exaggerated cases of chronic venous engorgement of the liver (*vide* p. 96). Kelly, on the other hand, groups together under the heading of "multiple serositis" these cases of pericarditic pseudo-cirrhosis and the cases of "iced liver," or chronic universal perihepatitis, and while admitting some anatomical distinctions, regards them as very much alike clinically.

(II) *Chronic Perihepatitis associated with Arteriosclerosis and Granular Kidneys.*—Chronic perihepatitis is frequently associated with arteriosclerosis and granular kidneys; this was so in 19 out of Hale White's[4] 22 cases. In this group there is not the same intimate relationship between chronic perihepatitis and thoracic lesions as in the previous category, though the two conditions may be found together.

There appears to be some connexion between arteriosclerosis and fibrosis of the serous membranes (perivisceritis[5]). It is conceivable that arteriosclerotic change in the kidneys is a disposing cause of chronic inflammation in the body generally. As Flexner[6] has shewn, the bactericidal power of the blood is reduced in chronic renal disease and so allows microbic infections to occur. It is reasonable to believe that chronic perihepatitis and peritonitis might be produced by micro-organisms of no great virulence but capable of inducing considerable fibrosis. It is more probable that the process is microbic than that it is purely toxic and due to an altered (uraemic) condition of the blood. In either case the influence of arteriosclerosis and granular kidneys should lead to a similar change in the other serous cavities, the pleurae and pericardium. This condition of combined inflammation of several serous membranes—polyorrhymenitis[1] or multiple serositis—sometimes occurs in cases of granular (arteriosclerotic) kidneys, but it is by no means constant to find another serous membrane involved as well as the peritoneum. In order to explain cases in which the peritoneum alone is attacked or is much more affected than any of the other serous membranes, it may be suggested that the facilities for infection are greater in the abdomen and that the resistance of the peritoneum has been specially lowered in these cases. It has been thought that arteriosclerosis of the small vessels of the peritoneum may produce chronic peritonitis—a fibrosis due to impaired nutrition.

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[1] Gilbert et Garnier. *Compt. rend. Soc. Biol.*, Paris, 1898, l, 48. /l.c
[2] Heidemann. *Berlin. klin. Wchnschr.*, 1897, xxxiv, 92.
[3] Pick. *Ztschr. f. klin. Med.*, Berlin, 1896, xxix, 385.
[4] Hale White. *Trans. Clin. Soc.*, Lond., 1888, xxi, 221.
[5] Labadie-Lagrave et Deguy. *Arch. gén. de méd.*, 1898, ii, 411 (Perivisceritis).
[6] Flexner. *Journ. Exper. Med.*, N.Y., 1896, i, 559.

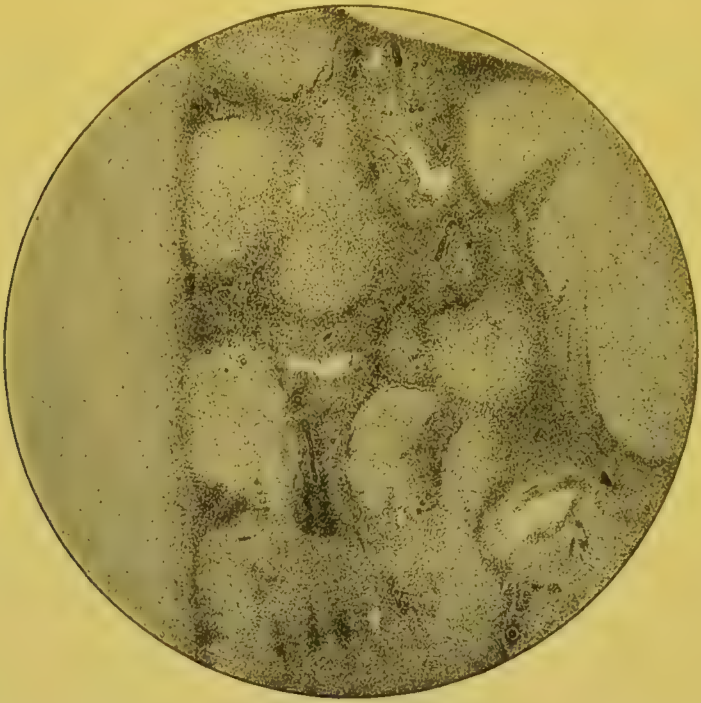

FIG. 23.—Microscopic section shewing homogeneous hyaline membrane on the surface of the liver with a similar hyaline change in the fibrous tissue of the portal spaces in the underlying liver substance. From a case of myxoedema.

As an example of this form the following case may be quoted: A woman aged sixty-nine years had ascites requiring paracentesis; eventually she died in

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[1] For an account of this condition see Taylor, F., *Brit. Med. Journ.*, 1900, ii, 1698; Kelly, A. O. J., *Am. Journ. Med. Sc.*, 1903, cxxv, 116.

Recently Letulle has described
Chronic syphilitic peritonitis

Letulle. Presse méd., Paris 1918, 477

an extremely thin and cachectic state. The necropsy revealed granular kidneys, universal chronic peritonitis, a liver which weighed only 23 ounces, and on section looked cirrhotic, but not in the least nutmeggy. Microscopically the liver was nutmeggy and shewed only slight replacement fibrosis. The heart, 8 ounces, was devoid of epicardial fat ; on section it shewed brown atrophy of the myocardium.

A well-marked example of chronic universal perihepatitis with similar changes in the pleurae and pericardium, and granular kidneys, occurred in a woman who died in St. George's Hospital in coma in 1898. She had often been treated for myxoedema. She had ascites, universal chronic perihepatitis, perisplenitis, and peritonitis with a number of fibrous nodules in the peritoneum near the umbilicus (peritonitis fibrosa). Both layers of the pericardium were much thickened, and the cavity contained excess of fluid. The pleurae were both thickened, shewed adhesions, and contained about 8 ounces each of clear fluid. The liver (60 ounces) was nutmeggy and free from cirrhosis. Microscopically there was a dense layer of fibrous tissue on the surface of the liver replacing the capsule. There was subcapsular atrophy of the liver cells, while the branches of the hepatic artery and portal vein appeared as prominent objects from a very peculiar swelling of their coats (*vide* Fig. 23). This change seemed to be due to myxomatous degeneration and to be connected with the primary disease—myxoedema. The kidneys (right, $3\frac{1}{2}$ ounces ; left, $3\frac{1}{2}$ ounces) were red and granular. The heart weighed 14 ounces. The thymus could not be found, and the pituitary was not enlarged. The thyroid was atrophied and pale yellow in colour. There was obsolete tubercle in the lungs.

(III) *Universal Chronic Perihepatitis due to other or Obscure Causes.*— In a few instances perihepatitis may be associated with syphilitic lesions of the liver, but, as a rule, hepatic gummas only give rise to a local thickening of the capsule of the liver.

Cheadle[1] believed that perihepatitis is more marked and frequent in association with syphilitic disease of the liver than in any other condition. In 22 cases of universal perihepatitis collected by Hale White[2] there were 3 in which syphilis was the apparent factor. Numerous small syphilitic gummas may so extensively involve the capsule as to set up universal perihepatitis (Sharkey,[3] N. Moore[4]).∧

In the following case universal perihepatitis and chronic peritonitis were associated with the presence of three old and several recent gummas in the liver. The patient, a man aged thirty-seven years, who denied syphilis, but drank 3 pints of beer daily, had been well until three and a half months before his death, when he began to suffer from morning sickness and abdominal pain. A month later he became jaundiced, ascitic, oedematous as to his legs, and vomited a little blood. He was tapped twice, 40 pints being removed. The urine was free from albumin. After death universal chronic perihepatitis and a moderate degree of general chronic peritonitis were found ; the liver weighed 56 ounces and contained three gummas in the right lobe and several scars and recent gummas in the left lobe; there was no general fibrosis of the liver. There appeared to be kinking of the portal vein and bile-duct in the portal fissure, but on cutting away the adhesions around them the vein and duct were found to be unobstructed. The kidneys weighed 6 ounces each and were healthy and free from lardaceous change. The heart weighed 8 ounces and was healthy.

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[1] Cheadle, W. B. *Some Cirrhoses of the Liver*, pp. 41, 43, 1900.
[2] Hale White. *Trans. Clin. Soc.*, Lond., 1888, xxi, 219.
[3] Sharkey, S. J. *Trans. Path. Soc.*, Lond., 1883, xxxiv, 118.
[4] Moore, N. *Ibid.*, 133.

In rare instances universal chronic perihepatitis may be associated with intra-abdominal malignant disease or be due to tuberculosis. In the latter case the lesions are those of chronic fibrosis and are very different from those of ordinary tuberculous peritonitis.

Chronic peritonitis and perihepatitis may be associated with and possibly due to the condition sometimes called "cirrhosis of the stomach," though really diffuse carcinoma of the organ. I have seen two cases of universal perihepatitis associated with this condition.

It has been thought to be due to traumatic peritonitis, for example as the result of the kick of a horse (Gazzotti [1]). It may also be found in cases of backward pressure without any adherent pericardium.

A woman, aged thirty-two at the time of her death, was very frequently under my care with chronic bronchitis, cyanosis, and recurring ascites, for which she was tapped forty-one times, over five hundred pints of fluid being removed. At the necropsy the kidneys, pericardium, and left side of the heart were normal; the lungs shewed chronic bronchitis and some emphysema, and the right side of the heart was dilated. There was marked chronic peritonitis in the upper part of the abdomen involving the capsules of the liver and spleen.

In other instances alcoholism has been thought to be the only antecedent condition, while sometimes, as in the very extreme condition—systematic hypertrophic cirrhosis of the peritoneum described by Du Pacquier [2]—no cause is forthcoming.

It is conceivable that in some instances chronic perihepatitis and peritonitis may start from foci of local irritation which usually only give rise to circumscribed perihepatitis. In such cases the widespread effect is analogous to the development of a cheloid in the skin after a slight injury.

Nicholls recorded in full detail a case of widespread chronic hyperplastic inflammation of the serous membranes (including perihepatitis) in a woman aged forty-eight with a chronic duodenal ulcer and slight chronic interstitial nephritis. The changes in the serous membranes were referred to irritation and possibly infection from the duodenal ulcer.

Finally, since the process is evidently a chronic and progressive inflammation, it is reasonable to imagine that it is due to infection with micro-organisms of a somewhat attenuated virulence. Nicholls suggests *Bacillus tuberculosis*, *B. coli*, and *B. typhosus* as probable organisms; this

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[1] Gazzotti. *Policlin.*, Roma, 1909, xvi (Sez. med.), 381.

[2] Du Pacquier. *Arch. gén. de méd.*, 1897, clxxx, 651.

suggestion is extremely attractive, but proof is wanted. The occurrence of a special form of reaction to the tubercle bacillus in the intestines—chronic hyperplastic tuberculosis—makes it tempting to suppose that attenuated human tubercle bacilli or possibly bovine bacilli (M'Weeney[1]) may be the cause of at least some cases of chronic perihepatitis and peritonitis.

Morbid Anatomy.—The surface of the liver is covered over by a crust or coating of fibrous tissue of cartilaginous consistence. It is glistening and white, resembles the icing of confectioners, and thus accounts for the name "sugar-iced liver" (*Zuckergussleber*) which has been applied to this condition by Curschmann. The surface of this casing is pitted or fenestrated, probably as a result of rupture during its spontaneous shrinking and contraction. This coat can be peeled off, leaving the peritoneal surface in a fairly healthy state. The liver is compressed and deformed by the constant traction which the cicatricial tissue exerts upon it. Thus the anterior margin may be bent upwards and over so as to touch the convex surface. The convexity of the liver is usually much more affected than the under surface, and of course the portions uncovered by peritoneum are free from the change. In uncomplicated cases the gall-bladder is nearly always collapsed and buried under thick membrane and is with difficulty discovered, its position being sometimes merely indicated by a depression.

In the one instance in which I have seen it dilated there was malignant disease of the peritoneum in addition to chronic perihepatitis and peritonitis.

Even when the portal fissure is invaded by the perihepatitis the portal vein hardly ever becomes compressed as might be expected. Hale White[2] points out that if the portal vein were compressed or kinked the bile-duct would also be affected, and that this must be very rare from the clinical absence of jaundice. His point is that ascites is due to the chronic peritonitis and not to mechanical interference with the portal vein, for in two cases—very exceptional ones—of universal chronic perihepatitis without any chronic peritonitis there was no ascites. In the case given on pages 165, 166, however, there appeared to be kinking of the portal vein and bile-duct. There may be adhesions between the surface of the liver and the diaphragm, abdominal walls, and adjacent organs.

The *liver* is usually soft, fatty, and shews chronic venous engorgement with general atrophy. Multilobular cirrhosis is very rarely combined with well-marked perihepatitis of the kind now under discussion. It is true that adhesions and opacity with thickening of the capsule are often seen in advanced cirrhosis, but the combination of the "iced liver" and cirrhosis is so rare that in Nicholls' monograph one example only is admitted. Superficial fibrosis spreading in from the surface (Glissonian cirrhosis) is not uncommon, but it does not extend for any distance.

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[1] M'Weeney. *Trans. Roy. Acad. Med. Ireland,* 1906, xxiv, 402.

[2] Hale White. Allbutt's *System of Medicine,* 1897, iv, 120.

Microscopically the thickened membrane is seen to lie on Glisson's capsule, which is thrown into wavy folds. This "icing" membrane is composed of well-formed fibrous tissue arranged in horizontal laminae with a few nuclei between them. Oertel[1] describes extensive involvement of the superficial lymphatics. The structure is like that of a lamellar fibroma of the spleen. The fibrous tissue has undergone hyaline degeneration. From the presence of this change Nicholls speaks of the condition as hyaloserositis. The membrane does not contain blood-vessels and there is usually a distinct line of separation between the membrane and the underlying capsule of the liver. In the deeper layers there are collections of leucocytes and mast-cells; there is no reaction for fibrin (Nicholls). Carnot and Amet[2] describe a thin wavy layer of elastic tissue, containing numerous small blood-vessels.

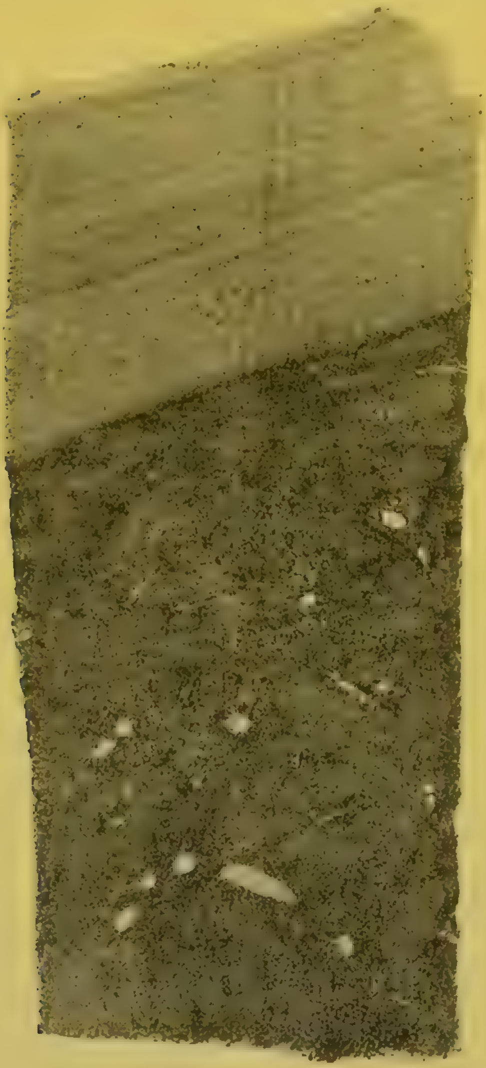

FIG. 24.—The thick "icing" in chronic universal perihepatitis. The line of distinction between it and the liver is well seen. The liver shewed some fibrosis and chronic venous engorgement. (Photomicrograph by Dr. H. Spitta.)

The condition of the underlying liver varies a little; it is rarely absolutely normal, being generally atrophied, with fatty and pigmentary changes in the liver cells; chronic venous engorgement is very common. Not very rarely there is slight Glissonian cirrhosis or fibrosis spreading a short way into the substance of the liver. The underlying liver tissue may shew a similar hyaline change in the fibrous septa (Fig. 23).

In cases of calcified adherent pericardium cirrhosis of the liver has been described (*vide* p. 98; Diemar,[3] Wells[4]).

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[1] Oertel. *Arch. Int. Med.*, Chicago, 1908, i, 385.

[2] Carnot et Amet. *Arch. de méd. expér. et d'anat. path.*, Paris, 1906, xviii, 758.

[3] Diemar. *Ztschr. f. Heilk.*, 1899, xx, 257.

[4] Wells. *Am. Journ. Med. Sc.*, Phila., 1902, cxxiii, 259.

The *spleen* is usually rather larger than natural, its capsule shares in the chronic thickening of the peritoneum, and is often firmly adherent to the diaphragm and abdominal wall.

The *kidneys* in cases associated with indurative mediastino-pericarditis, adherent pericardium, and chronic pleurisy may shew little change except chronic venous engorgement. When chronic perihepatitis is associated with arteriosclerosis, the kidneys usually share in the change and are red and granular.

The *peritoneum* shews the same fibroid and hyaline change as the capsule of the liver, and undergoes the same cicatricial contraction and puckering. The great omentum is rolled up and transformed into a hard cord, the mesentery is thickened, and by its retraction tethers the intestines, which are much shortened, to the spine, so that they cannot reach to the front of the abdominal wall. As a result of this the abdomen may be uniformly dull in front. The right pleura is more often affected by the chronic fibroid change than the left. The body as a whole is thin and may be extensively oedematous.

Clinical Picture.—*Sex.*—The sexes are about equally affected, thus contrasting with cirrhosis, in which the male sex predominates.

In Hale White's[1] 21 cases there were 13 males and 8 females. In 20 cases that I have tabulated the sexes were equally affected.

Age.—The age varies considerably, but is generally about the same as in cirrhosis.

In Hale White's cases the average age was forty-seven and a half years, the youngest being twenty-nine and the oldest sixty-eight years. In my 20 cases the average age was 42·6 years, being practically the same in the two sexes; the extremes were fourteen and sixty-eight years.

Heredity does not appear to have any influence in the development of the disease.

The *onset* of ascites is usually gradual, but in some instances is acute; this was so in 6 out of 14 cases selected by Nicholls. The condition has been thought to date back to, and be started by, an acute inflammatory change involving the pericardium or liver.

The *course* of the disease is slow and extends over years. Nicholls finds that the duration of the disease is from two to sixteen years. Ascites, which constantly recurs, is the main symptom, but the general health and strength are fairly maintained for a long time. Emaciation gradually appears and death occurs from some intercurrent disease or infection, such as acute peritonitis or pneumonia.

In one case under my care death was due to influenza, and at the necropsy there were two small recent ulcers in the first part of the duodenum with acute inflammation of the duodenum.

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[1] Hale White. Allbutt's *System of Medicine*, 1897, iv, 121.

Signs and Symptoms.—The condition is practically always accompanied by ascites, and thus contrasts with cirrhosis which may be quite latent. Ascites must be due to chronic inflammation where the process is spreading, for it is difficult to imagine that any exudation could occur from the extremely thickened membrane seen in the oldest parts of the change. Ascites is the chief physical sign and requires frequent tapping. Thus, Osler[1] refers to a child in whom this operation was performed 121 times. Tapping becomes necessary at shorter intervals and may be called for every fortnight or even sooner. Ascites is compatible with fair health and strength during the earlier part of the disease, and in the intervals between the tappings the patient may be up and about.

A woman with universal chronic perihepatitis, secondary to calcification of the pericardium, who died after her fifty-second tapping, was married after being tapped twenty times. She was under the care of Dr. Ewart, in St. George's Hospital.

The recurrence of ascites combined with fair health, or at any rate freedom from toxaemia, are important points in the diagnosis from cirrhosis. The ascitic fluid is clear and straw-coloured, but contains a high percentage of albumin (3 per cent), which allies it with inflammatory exudations rather than with passive transudations. If allowed to stand, threads of fibrin may form. Occasionally the ascites is encysted by adhesions. The liver may be enlarged and palpable in the earlier stages, but becomes smaller as time goes on. Ascitic distension of the abdomen may interfere with the estimation of the size of the liver and spleen. Some enlargement of the spleen is usual in young subjects.

The omentum may be felt as a transverse band passing across the abdomen, and must not be regarded as the lower border of the liver. The subcutaneous abdominal veins (superior and inferior epigastric veins) are sometimes enlarged and are seen to form a collateral circulation between the superior and inferior venae cavae. These veins do not converge towards the umbilicus, as is the case in the collateral circulation of portal obstruction. Jaundice is almost always absent; it was present in only one of Hale White's 22 cases, and when it occurs is probably due to some independent factor. In a case of ascites and intense jaundice examined by Bryant[2] the ascites was due to perihepatitis, and the jaundice to acute yellow atrophy. The *urine* is diminished in amount and may contain albumin, either from chronic venous engorgement or from arteriosclerotic change in the kidneys.

In addition to ascites there may be signs of the conditions specially associated with chronic perihepatitis: viz. (i) adherent pericardium, chronic mediastinitis, dense pleural adhesions, or (ii) arteriosclerosis and granular kidney. Oedema of the legs is common late in the disease and may extend to the trunk and upper extremities.

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[1] Osler. *Practice of Medicine*, p. 576, 4th ed., 1901.

[2] Bryant, J. H. *Guy's Hosp. Gaz.*, 1900, xiv, 141.

Since the liver substance is well preserved, there is little constitutional disturbance and no tendency to hepatic inadequacy or to haemorrhages, drowsiness, and the other toxaemic manifestations seen in cirrhosis. If such symptoms do develop, they are to be referred to concomitant renal disease. In many cases which clinically present themselves as failing heart from granular kidneys, with ascites and general dropsy, there is universal perihepatitis. There may be loss of appetite and dyspepsia, and the relaxed and flabby condition of the abdominal walls favours constipation. Gastro-intestinal haemorrhage does not occur unless there is some complication, such as gastric or duodenal ulcer or, in rare instances, cirrhosis.

Diagnosis.—The presence of long-continued ascites not manifestly due to obstructive heart or lung disease, or to chronic nephritis, points very strongly to universal chronic perihepatitis and peritonitis.

Differential Diagnosis.—*From Cirrhosis of the Liver.*—When ascites occurs in cirrhosis the end is usually near and paracentesis is not likely to be often required unless the ascites is due to associated chronic peritonitis. Repeated tappings are therefore strongly in favour of chronic peritonitis and perihepatitis; evidence of arteriosclerotic kidney disease would also strengthen this view. Jaundice, grave constitutional and toxaemic symptoms, haematemesis, melaena, haemorrhages, and an enlarged liver and spleen point to cirrhosis.

From the effects of backward pressure due to dilatation of the right side of the heart, mitral disease, etc., the diagnosis depends on the recognition of the underlying cause and on the good effects of treatment by cardiac tonics. In cases of adherent pericardium and advanced nutmeg liver (compare p. 96) the resemblance to chronic perihepatitis associated with adherent pericardium is very close, but cardiac treatment is much more effective in the former condition, which occurs mainly in the young, while universal chronic perihepatitis is little affected by this treatment and usually occurs in adults.

In malignant disease of the liver with ascites there is progressive enlargement of the liver, with perhaps some palpable nodules of new-growth, and the course of the disease is much more rapid.

Syphilitic gummas in the liver may closely imitate perihepatitis by giving rise to recurrent ascites, and the distinction between them is often very difficult. Outward signs of syphilis may be entirely absent, even when there are numerous gummas in the liver. The Wassermann test should be tried; the best means of making a diagnosis is vigorous anti-syphilitic treatment.

Chronic Tuberculous Peritonitis.—In this condition the effusion is seldom very large and lumps may be felt in the abdomen. Induration around the umbilicus, concomitant pleurisy, and fever are points in favour of tuberculous peritonitis. In cases of doubt the tuberculin tests can be employed.

Prognosis.—The prognosis is most unfavourable: death always occurs eventually, though it may be postponed for a long period. According to

Kelly[1] 50 per cent of the patients live four years or more, and 70 per cent two years or more. Thus a patient may live to be tapped 60 or even 100 times. But the tappings become necessary at shorter intervals, and the patient gradually gets weaker. The disease has been said to be more rapidly fatal in cases associated with adherent pericardium.

Treatment.—Treatment is only palliative. Dry diet, viz. limiting the intake of fluid, and diuretics, such as citrate of caffeine, diuretin, strophanthus, digitalis, apocynum, may be tried. Iodide of potassium is usually given a trial, but it is very doubtful if it has any effect except when there is syphilitic disease of the liver. However, as the diagnosis between gummas involving the portal vein and universal perihepatitis may be difficult, it is well to give iodides on the chance of an error in diagnosis. Paracentesis should be performed when necessary; a permanent drain externally is dangerous from the risk of infection and subsequent peritonitis, and it should therefore not be employed; but the peritoneum may be drained into the subcutaneous tissues as in the ascites of cirrhosis (*vide* p. 260).

Removal of a small piece of the membrane covering the liver and union of the liver with the abdominal wall, as in the Talma-Morison operation for cirrhosis, tried in one case of Dr. Ewart's[2] in St. George's Hospital, and I believe by others, failed to give any permanent benefit.

CIRRHOSIS OF THE LIVER

Classification of the Various Forms of Cirrhosis

Cirrhosis of the liver has been classified in various ways, according to its causes, according to the methods by which the cirrhosis is brought about, according to the anatomical changes and lesions, and according to the distinctive clinical features. Some writers, such as Lancereaux,[3] consider cirrhosis under the headings of alcoholic, malarial, and syphilitic. But as other factors may lead to cirrhosis, this classification is not sufficiently broad.

Classification according to the Methods by which Cirrhosis is Induced.—The following is Chauffard's[4] classification:—

I. Vascular. (*a*) Toxic.
    (i) Poisons taken by the mouth.
    (ii) Poisons manufactured in the intestinal tract.
  (*b*) Infective.
    (i) The direct action of micro-organisms.

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[1] Kelly, A. O. J. *System of Medicine* (Osler and M'Crae), 1908, v, 742.
[2] Ewart. *Brit. Med. Journ.*, 1899, i, 908.
[3] Lancereaux. *Traité des maladies du foie et du pancréas*, 1899.
[4] Chauffard, A. *Traité de médecine* (Bouchard, Brissaud), 1902, v, 172.

- (ii) The action of bacterial toxins, either produced by bacteria in the liver itself or manufactured elsewhere in the body and carried to the liver.
- (c) Dystrophic.
  - (i) Due to arteriosclerosis; this form of cirrhosis would be homologous to a granular arteriosclerotic kidney, the fibrosis being a replacement fibrosis.
  - (ii) Due to chronic venous engorgement, the fibrosis of a nutmeg liver.

II. Biliary. (a) Secondary to obstruction of the larger bile-ducts.
- (b) Primarily an inflammation of the smallest bile-ducts, leading to a unilobular cirrhosis. Hanot's hypertrophic cirrhosis with chronic jaundice.

III. Capsular cirrhosis or perihepatitis.
- (a) Chronic localised.
- (b) Chronic universal.

This classification is too elaborate, for the toxic and infective forms overlap, and from a clinical standpoint some of these subdivisions are too minute to be of practical value.

Anatomical Classifications.—Adami[1] has proposed a division of cirrhosis on anatomical grounds.

(I) Portal cirrhosis, (a) with enlargement, (b) with contraction.
(II) True biliary cirrhosis.
(III) Obstructive biliary cirrhosis, (a) with enlargement, (b) with contraction.
(IV) Pericellular or diffuse cirrhosis.
(V) Capsular cirrhosis (cirrhosis with perihepatitis).

A certain amount of fibrosis may spread into the liver substance from the capsule, but the most marked perihepatitis may exist without any trace of hepatic fibrosis; when an extension inwards does occur, the sequence of events is analogous to what occurs in interstitial pneumonia secondary to chronic pleurisy (pleurogenous pneumonia). It has been suggested that toxic bodies manufactured in the process of perihepatitis may be absorbed by the lymphatics, pass into the liver, and set up fibrosis.

(VI) Senile atrophy and arteriosclerosis.

Arteriosclerotic fibrosis of the liver might naturally be expected in the aged, and a statement to this effect is sometimes made. Personally, from a microscopic examination of a large number of livers I consider it to be very rare, and when present of very slight degree. I have often seen considerable atrophy of the liver cells without any replacement fibrosis. Barbacci,[2] by means of

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[1] Adami, J. G. *Principles of Pathology*, 1911, ii, 471.
[2] Barbacci. *Sperimentale*, Firenze, 1910, lxiv, 31.

Bielschowski's silver nitrate method, has found a diffuse fibrosis which is not shewn by ordinary staining methods.

(VII) Cirrhosis from chronic venous engorgement. This question is discussed on p. 88.

The Author's Anatomical Classification.—A purely structural classification may be drawn up from the microscopic appearances as follows:—

(I) *Multilobular cirrhosis* in which a varying number of lobules are enclosed in a fibrous ring; the following sub-varieties may be recognised:—

- (*a*) With fatty change in the liver cells.
- (*b*) With hyperplasia of the liver cells to such an extent that nodular cirrhosis, or cirrhosis with adenoma, results.
- (*c*) With pigmentation of the liver cells and also of the fibrous tissues (haemochromatosis).
- (*d*) Mixed, with intercellular infiltration of the hepatic lobules. This is sometimes seen in rapidly advancing cirrhosis.

Multilobular cirrhosis is almost always due to poisons brought by the portal vein, and is therefore spoken of as periportal or venous cirrhosis, but it may, in exceptional instances, be associated with changes in the larger bile-ducts.

(II) *Unilobular cirrhosis*; each lobule is separated from its fellows by a delicate fibrosis. The arrangement thus far imitates that normally seen in the pig's liver. Unilobular cirrhosis is specially related to inflammation of the minute bile-ducts, but is sometimes described as a result of cholangitis of the larger ducts. Unilobular cirrhosis is often complicated by multilobular cirrhosis and is frequently associated with intercellular cirrhosis, so that a mixed form of cirrhosis results.

(III) *Intercellular cirrhosis*; each hepatic cell is separated by young connective tissue from its fellows.

(IV) *Mixed cirrhosis*; in which there are various combinations of the three preceding main types, the multilobular, unilobular, and intercellular. This is met with in many cases of portal cirrhosis, especially when the course of the disease has been rapid, and is also seen at the termination of long-standing cases of hypertrophic biliary cirrhosis. Mixed cirrhosis is a common anatomical form, but has no claims to be considered as a clinical type of cirrhosis.

(V) *Sporadic fibrosis*, in which there are isolated patches of fibrosis not corresponding with any of the preceding categories. Under this heading come local fibrosis around parasites, cysts, granulomas, the results of small areas of focal necrosis, and the replacement fibrosis seen in chronic venous engorgement.

In the foregoing anatomical classifications the portal and biliary forms of cirrhosis are of great clinical importance, the others, with the exception of the intercellular cirrhosis of congenital syphilis and the cirrhosis

of haemochromatosis (p. 303), are chiefly of pathological interest. Oertel[1] considers that a classification of cirrhosis is not possible on anatomical grounds, because all cirrhosis is intralobular and the described forms merge freely into one another.

Clinical Classifications.—If any formal classification be adopted, it should be one that can be used at the bedside. A large number of different varieties of hepatic cirrhosis have been described, and in many cases the probable causes, or associated conditions, of the cirrhosis, such as alcohol, malaria, syphilis, tuberculosis, diabetes, dyspepsia, biliary obstruction, have been taken as grounds for establishing different types of the disease. Again, cirrhosis has been classified according to the size of the liver or of the spleen. A large (hypertrophic) cirrhotic liver and a small (atrophic) cirrhotic liver are often spoken of; while the French school has distinguished different types of biliary cirrhosis not only according to the relation in size between the liver and spleen, but also according to the time incidence of enlargement of the two organs. Thus, the liver may be much larger (hepatomegalic), of relatively much the same size (splenomegalic), or even smaller (hypersplenomegalic biliary cirrhosis) than the spleen; or the liver may be enlarged before (presplenomegalic), at the same time (splenomegalic), or after (metasplenomegalic hypertrophic biliary cirrhosis) the enlargement of the spleen is detected. These titles serve to shew the minute and elaborate character of the classification attempted.

Senator's classification can to some extent be utilised in clinical practice.

I. Portal Cirrhosis.
  (*a*) The hobnailed liver, diminished in size.
  (*b*) With jaundice, which is either due to gastro-intestinal catarrh obstructing the common bile-duct, or to pressure exerted on the intrahepatic radicles of the bile-ducts.
  (*c*) With increased size of the liver, which may subsequently be followed by a diminution in bulk.
II. Biliary Cirrhosis.
  (*a*) Due to calculous obstruction of the ducts. No splenic enlargement, no ascites.
  (*b*) With an enlarged spleen. This is a transitional form to the next.
III. Hypertrophic biliary cirrhosis with chronic jaundice.

From a practical point of view it seems to me to be best to make, in the first instance, a broad division of cirrhosis into two principal types as shewn by their clinical features:—

(*a*) Ordinary or common cirrhosis (portal),

in which haematemesis is an early symptom and ascites a terminal incident. Jaundice is not prominent; death may be preceded by a toxaemic state without much or any ascites.

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[1] Oertel. *Arch. Int. Med.*, Chicago, 1908, i, 390.

The liver may be either large or small, but shews multilobular fibrosis; the spleen is enlarged, but not so constantly or markedly as in the second category.

(*b*) The biliary type. Jaundice is the most prominent feature and may be constant for long periods ; haematemesis and ascites are only met with exceptionally. A little ascites may develop as a terminal phenomenon shortly before death. The liver is enlarged, sometimes to an extreme degree. The surface is smooth and the fibrosis of a mixed, diffuse, or unilobular type. The spleen is enlarged, sometimes greatly.

In this work these two main types of cirrhosis will be adhered to, but short sections on the sub-varieties of cirrhosis, included under these two main types, will be added.

Portal Cirrhosis.
  Variety : Pigmentary Cirrhosis.
Biliary Cirrhosis.
  (i) Hypertrophic Biliary Cirrhosis.
  (ii) Obstructive Biliary Cirrhosis.

Comparison of the Main Forms of Cirrhosis with some Forms of Kidney Disease.—Portal cirrhosis may be compared to granular kidney. Hypertrophic biliary cirrhosis may be likened to chronic parenchymatous nephritis, the changes being due to poisons reaching the organs by the arterial blood-stream. The terminal production of multilobular cirrhosis in long-standing hypertrophic biliary cirrhosis corresponds to the evolution of a contracting or small white kidney, due to the addition of the changes of a granular or arteriosclerotic kidney to chronic parenchymatous nephritis (large white kidney). Lastly, obstructive biliary cirrhosis is comparable to consecutive nephritis.

PORTAL CIRRHOSIS

Synonyms: Common, Alcoholic, or Atrophic Cirrhosis; Multilobular, Annular, or Venous Cirrhosis; Laennec's Cirrhosis; Hobnailed, Gin- or Whisky-drinker's Liver; Chronic Interstitial Hepatitis.

THE term atrophic cirrhosis, though commonly used as synonymous with portal cirrhosis, is unsuitable, inasmuch as the liver is often larger than natural. The adjectives portal, multilobular, and common are more accurate. Old names, now forgotten, for the condition are granulated, tuberculated, lobulated liver.

Definition.—The disease is characterised anatomically by fibrosis spreading from the portal spaces and enclosing varying numbers of lobules, in which the cells tend to shew degenerative changes. Clinically dyspepsia, ~~haematemesis~~ gastro-intestinal haemorrhage, splenic enlargement, terminal ascites, and oedema of the feet are the essential features, while jaundice is either absent or transient and slight. The disease is commonest about the age of fifty and is frequently related to chronic alcoholism.

History.—The hardened condition of the liver was described by Vesalius, Harvey, Morgagni, and others. Payne[1] in reviewing the history of cirrhosis reproduced the drawing of a cirrhotic liver described in the *Philosophical Transactions* of 1685 by John Browne, surgeon to St. Thomas's Hospital. The ascites in this case, as in others before this date, was thought to be the result of drinking too much water, "a fault which," according to Payne, "the bold spirits of the time were much on their guard against." Matthew Baillie[2] gave a good naked-eye description of the disease under the name of "common tubercle of the liver"; he was really the first to separate it from "scirrhous tumours," and he regarded it as a disease peculiar to the liver and more apt to occur in those accustomed to drink spirituous liquors. The word "cirrhosis" was invented in 1819 by Laennec,[3] who regarded the bile-stained "hobnails" as masses of yellow (κιρρός) new-growth, invading the liver, hence cirrhosis. Hanot dutifully proposed that portal cirrhosis should be spoken of as Laennec's cirrhosis.

Carswell[4] in 1838 was the first to point out the presence and importance of fibrosis in cirrhosis, and was confirmed by Kiernan's histological researches. For further details as to the history of cirrhosis the reader is referred to Wickham Legg's[5] essay.

Etiology.—*Age.*—Portal cirrhosis is a disease of late middle life, and usually proves fatal about fifty years of age.

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[1] Payne, J. F. *Trans. Path. Soc.*, Lond., 1889, xl, 310.
[2] Baillie, M. *Morbid Anatomy*, p. 141, 1793.
[3] Laennec. *Traité de l'auscultation méd.*, 1819, tome i, p. 368 (*note*).
[4] Carswell. *Illustrations of Elementary Forms of Disease.*
[5] Legg, W. *St. Barth. Hosp. Rep.*, 1872, vii, 74.

In 165 adults whose livers were cirrhotic the average age was 48·7 years; of these, 121 were males (average age, 49·4 years) and 44 females (average age, 47 years). In Yeld's[1] 128 cases the average age was 47·5 years.

There is a definite group of cases of genuine portal cirrhosis in young children, which will be specially referred to elsewhere (*vide* p. 334).

Hilton Fagge[2] found that the average age of persons in whose bodies cirrhosis of the liver was discovered without any marked symptoms during life was five years higher than that of persons dying of the disease. This would suggest that the compensatory processes had been very successful; but in my own figures there is very little difference between these two classes.

In 78 patients dying from cirrhosis the average age was 48·3 years, whereas in 87 patients who died from independent causes but whose livers were cirrhotic the average age was 49·1 years.

There is reason to think that cases of cirrhosis with a very distinct alcoholic history are fatal at an earlier age than cases of cirrhosis in which alcoholic excess either did not exist or is not a prominent recent feature in the history. This is especially marked in the case of women.

In 114 cases tabulated by Fenton and myself the average age of all the alcoholic cases of cirrhosis was 46·6 years, as against 49·9 years for the non-alcoholic cases. Taking the ages of the sexes separately, the figures read as follows—alcoholic males, 47·8 years; non-alcoholic males, 49·3; alcoholic females, 42·8; non-alcoholic females, 51·5.

Sex.—Portal cirrhosis is commoner in men than in women; probably in the proportion of about 3 to 1.

Among 645 cases in which cirrhosis of the liver was found after death, obtained by adding together the statistics of Price (Guy's Hospital), Kelynack (Manchester), Yeld (St. Bartholomew's), Candler[3] (Charing Cross), and St. George's, there were 479, or 74·3 per cent, males, and 166, or 25·7 per cent, females. From the bills of mortality of New York for the years 1889–1899, Crook[4] found that of 4737 deaths from cirrhosis 2980, or 62·3 per cent, were males, and 1757, or 37·7 per cent, females. The Registrar-General's returns for England and Wales shew ~~much less difference~~; in the ten years ended 1904 there were certified as dying of cirrhosis 41,085 persons, of which 22,830, or 55·6 per cent, were males, and 18,255, or 44·4 per cent, females.

Variation
in the sexual
incidence

From statistics of St. George's Hospital it appears that cirrhosis is more often latent in men than in women (*vide* p. 227).

In children the incidence of portal cirrhosis is a little higher in the male sex; in 90 cases under twenty-one years of age there were 48

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[1] Yeld. *St. Barth. Hosp. Rep.*, 1898, xxxiv, 215.
[2] Hilton Fagge. *Guy's Hosp. Rep.*, 1874–5, xx, 193.
[3] Candler. *Arch. Neurol. Claybury Asylum*, 1907, iii, 439.
[4] Crook. *Med. News*, N.Y., 1902, lxxx, 246.

males and 42 females (Woolley[1]); in 67 cases of alcoholic cirrhosis in children 39 were in boys and 28 in girls (E. Jones[2]).

Occupation, etc.—Cirrhosis is more frequent in those who are brought in contact with alcoholic drinks, especially publicans, commercial travellers, and others who have occasion frequently to drink over business.

In 149 persons connected with the liquor traffic, Dickinson[3] found that post-mortem examination shewed cirrhosis to occur in 22, while in 149 other persons not brought specially into contact with alcohol it was present in 8 only.

Cirrhosis is more often seen in those whose life is sedentary than in persons leading an active outdoor life. Sir D. Duckworth[4] in 1874 quoted the experience of Sir W. Gull and Sir G. Burrows to the effect that cirrhosis was rare in the upper and well-to-do classes.

Geographical Distribution.—The distribution of cirrhosis does not run hand in hand with that of alcoholism. The abuse of alcoholic drinks is very widespread, but the distribution of cirrhosis does not by any means correspond. Thus, in hot countries alcoholic excess more often tends to produce a rapid reaction in the liver, such as hepatitis, while in cold climates cirrhosis is a more frequent result. But in temperate climates the incidence of cirrhosis varies very considerably; thus, cirrhosis is common in London, but comparatively rare in Scotland. There are also curious differences as to the distribution of cirrhosis in France which cannot be correlated with that of temperance and intemperance. It is an interesting question whether the incidence of cirrhosis has undergone any marked change in the course of years. In 1857 Budd[5] wrote that "cirrhosis is more common in England and Scotland than in France"; at the present day cirrhosis is probably quite as frequent in France as in England, and much more so than in Scotland. In this connexion it is significant that alcoholism has enormously increased in France of late years. In India cirrhosis is much commoner in natives than in Europeans; the reverse holds good as regards hepatic abscess. In England and Wales the death-rate from cirrhosis per million living has greatly increased since the Registrar-General reports were instituted; in the quinquennium 1866–70 it was 42, and there was an almost uniform increase up to 134·6 per million living in the quinquennium 1896–1900; in the quinquennium 1901–5 there was a fall to 121. As already mentioned, the death-rate from cirrhosis fluctuates with that of alcoholism in England and Wales.

Heredity.—Multilobular cirrhosis is not a "family" disease or one that tends to recur in members of the same family; in this way it contrasts with hypertrophic biliary cirrhosis, which may attack several members of the same family. In a few instances several adults may die of alcoholic cirrhosis, probably from a family failing towards alcoholism. In children the death of two or more members of the same family from multilobular cirrhosis is less rare. This may depend on hereditary influences, syphilis disposing the organ to the incidence of ordinary cirrhosis—parasyphilitic cirrhosis (*vide* p. 381). An important factor in the development of cirrhosis is the vitality of the liver; if its resistance is congenitally feeble, factors otherwise harmless, such as the specific fevers and indiscretions in diet, may induce cirrhosis. It is conceivable that alcoholism in the mother may so influence the livers of her offspring that cirrhosis develops in them more readily than it otherwise would. Precocious alcoholism or peculiarly unsuitable food, such as pickles or fish soaked in vinegar, may cause cirrhosis in two or more members of the same family. Jollye[1] described cirrhosis in a brother and sister accustomed to take vinegar.

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[1] Woolley. Unpublished thesis for M.D. Cantab., 1906.

[2] Jones, E. *Brit. Journ. Children's Diseases*, 1907, iv, 6.

[3] Dickinson, W. H. *Med.-Chir. Trans.*, 1873, lvi, 34.

[4] Duckworth. *St. Barth. Hosp. Rep.*, 1874, x, 56.

[5] Budd. *Diseases of the Liver*, p. 150, 3rd ed., 1857.

Two sisters, aged nine years and ten years, died in St. George's Hospital in 1899 and 1901 with hobnailed livers weighing 20 and 12 ounces respectively. In both of them laparotomy was performed: in the younger because tuberculous peritonitis was suspected; in the older, who had a temperature of 104° and diarrhoea, because perforation of a typhoid ulcer appeared probable. The mother was extremely alcoholic, but persisted that she had not given alcohol to the children. A brother aged sixteen years had laparotomy performed for supposed tuberculous peritonitis in St. Thomas's Hospital in 1901; no evidence of perihepatitis or peritonitis, but a hobnailed liver was found. All three cases died shortly after laparotomy and were examined after death. No abdominal lesion except hepatic cirrhosis and no evidence of syphilis was found in any of them.

Pathogeny.—Cirrhosis of the liver is the result of some poison, or possibly of poison-producing bodies such as micro-organisms, reaching the liver. These agents may reach the liver either (1) by the portal vein, or (2) by the hepatic artery; in other words, they may be derived from the alimentary canal or from the general circulation. When the active agent travels by the portal vein, the resulting cirrhosis is usually of the ordinary portal type; when the liver is affected secondarily to an arterial infection or toxaemia, the cirrhosis is often of a more mixed type, and may then resemble that of biliary cirrhosis. In some instances, however, in which the poison arrives by the hepatic artery, the cirrhosis is of the portal type.

In reviewing the etiology of cirrhosis it will be convenient, first of all, to consider the poisons and micro-organisms which reach the organ by the portal vein, and then those conveyed by the hepatic artery.

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[1] Jollye. *Brit. Med. Journ.*, 1892, ii, 858.

Goldzieher describes pluriglandular insufficiency as a
disposing factor to cirrhosis, but the changes in the
endocrine glands might be regarded as due to the
same factor as the cirrhosis

Goldzieher, M. Wien. med. Wchnschr., 1921, LXXI, 225 year 19[illegible]

In Chicago the death rate from
Cirrhosis appeared to fall after
the introduction of prohibition (Miller)

Stevenson, T. H. C. *Practitioner*, 1924, CXIII, 270

Miller. *Jour. Amer. Med. Assoc.*, Chicago
1921, LXXVI, 1646

SYNOPSIS

I.—POISONS CONVEYED TO THE LIVER BY THE PORTAL VEIN.
  (*a*) Ingested poisons : (1) alcohol ; (2) other poisons.
  (*b*) Poisons manufactured in the alimentary canal. Dyspeptic cirrhosis.
  (*c*) Umbilical vein cirrhosis and congenital syphilis.
  (*d*) Poisons manufactured in the spleen.
II.—MICRO-ORGANISMS CONVEYED TO THE LIVER BY THE PORTAL VEIN.
III.—POISONS CONVEYED TO THE LIVER BY THE HEPATIC ARTERY.
IV.—MICRO-ORGANISMS CONVEYED TO THE LIVER BY THE HEPATIC ARTERY.
CONCLUSION.

I. THE POISONS CONVEYED TO THE LIVER BY THE PORTAL VEIN may be divided into the following categories:—

(*a*) The poisons introduced into the intestinal tract from without: (1) alcohol, and (2) other bodies.

(*b*) Those manufactured in the alimentary canal as the result of faulty digestion, fermentation, and putrefaction.

(*c*) The poison of congenital syphilis, conveyed by the umbilical vein.

(*d*) Poisons manufactured in the spleen.

(*a*) INGESTED POISONS.—Of these, alcohol requires much the most consideration and discussion.

(1) *Alcohol and Alcoholic Drinks.*—With regard to the question whether or not *alcohol* is the cause of cirrhosis, clinical and experimental evidence are opposed. In practice there are few points more certain than that the abuse of alcoholic drinks is a frequent precursor of hepatic cirrhosis, while from experiments on animals the bulk of the evidence is in a contrary direction. The deaths from cirrhosis and from alcoholism, as shewn by the Registrar-General statistics,[1] correspond in a striking manner. Thus, in the ~~quinquennium 1871–75~~ there were ~~72~~ deaths from cirrhosis and ~~37~~ from alcoholism ~~per million living~~; in the ~~quinquennium 1901–5~~ the corresponding numbers were ~~121~~ and ~~78~~. year 19

Among drunkards, however, cirrhosis is not so frequent as is usually assumed. Thus in 250 necropsies on confirmed drunkards who died suddenly from the effects of alcohol, Formad[2] found cirrhosis in six only. The reason why alcoholics frequently escape cirrhosis is probably either that they have considerable resistance or that some other and necessary factor, which usually accompanies alcoholism, such as intestinal auto-intoxication, is wanting. Cirrhosis appears to be rare among the insane, and yet alcoholism is the cause of insanity in at least 10 per cent of the inmates of asylums (Sullivan); probably the explanation of the rarity of cirrhosis in alcoholic insanity is that only persons with an inherently stable nervous system can drink long enough to acquire advanced cirrhosis (Mott[1]).

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[1] ~~*Sixty-Eighth Annual Report of the Registrar-General for 1905*, p. 19~~.

[2] Formad. *Trans. Assoc. Amer. Phys.*, 1886, i, 225.

There are, of course, many cases of ordinary cirrhosis, especially in children, in which alcoholism can be excluded ~~as an antecedent factor in the production of cirrhosis~~.

Experimentally the introduction of alcohol into the stomach or portal vein of animals has usually given rise to fatty change and sometimes to some necrosis of the liver cells with a little small-celled infiltration around, but in only a few instances has cirrhosis resulted.

Numerous observers, Strassmann,[2] Afanasieff,[3] von Kahlden,[4] Sabourin,[5] Lafitte,[6] Pohl,[7] Scagliosi,[8] perseveringly administered alcohol in considerable quantities to animals over a fairly prolonged period, and entirely failed to detect any evidence of cirrhosis in the liver. Straus and Blocq[9] introduced a tube into the stomach of rabbits, and by this means gave 10 grams of alcohol daily; after three or four months a small-celled proliferation was found at the periphery of the lobules. This is hardly comparable to genuine cirrhosis, and, moreover, the introduction of the tube into the stomach complicates matters, inasmuch as it may have led to catarrh, and thus to the manufacture and subsequent absorption of other irritating bodies. De Rechter[10] has seen cirrhosis follow the continued administration of alcohol in dogs and rabbits. Out of 120 rabbits given alcohol experimentally for prolonged periods 4 shewed cirrhosis (Friedenwald[11]).

Since alcohol alone is not sufficient to account for cirrhosis either in man or animals, the undoubted association between alcoholism and cirrhosis must be explained in some other way. Two possibilities may be referred to: (i) That though ordinary ethylic alcohol itself does not produce cirrhosis, alcoholic drinks in virtue of other bodies contained in them are responsible for cirrhosis. It has been suggested that amylic alcohol is the important factor, but this requires definite proof. Lancereaux[12] believed that sulphate and bisulphate of potassium, which are added to wines and beers, are the causal factor in the production of cirrhosis, and supports his contention by the production of cirrhosis in rabbits, guinea-pigs, and dogs fed on sulphate of potassium. The amount of the salt given to these animals was very large, corresponding for an adult man, according to Vallin,[13] to a quantity of 60 to 350 grams per

[1] Mott. *Arch. Neurol. Claybury*, 1907, iii, 439.
[2] Strassmann. *Vrtljhr. f. gericht. Med.*, 1888, xlix, 232.
[3] Afanasieff. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1890, viii, 443.
[4] v. Kahlden. *Ibid.*, 1891, ix, 349.
[5] Sabourin. *La Gland biliaire d'Homme*, 1888.
[6] Lafitte, *Thèse de Paris*, 1892.
[7] Pohl. *Arch. f. exper. Path. u. Pharmak.*, 1893, xxxi, 281.
[8] Scagliosi. *Virchows Arch.*, 1896, cxlv, 546.
[9] Straus et Blocq. *Arch. de physiol. norm. et path.*, 1887, 3. s., x, 409.
[10] De Rechter. *Bull. Acad. Méd. de Belg.*, 1892, 4. s., vi, 425.
[11] Friedenwald. *Journ. Amer. Med. Assoc.*, 1905, xlv, 780.
[12] Lancereaux. *Bull. Acad. de Méd.*, Paris, 1897, xxxviii, 202; 1910, lxiii, 15.
[13] Vallin. *Ibid.*, 1897, xxxviii, 285, 343; 1898, xxxix, 257.

^ ; and cirrhotic livers are
occasionally found in animals
such as deer.

diem. Viola's[1] observations on cirrhosis in Venice lent support to Lancereaux's theory.

(ii) That alcohol and alcoholic drinks act indirectly, and either (*a*) set up gastro-intestinal catarrh and thus lead to the production of poisonous bodies which when carried to the liver cause cirrhosis, or (*b*) when acting on the liver simultaneously with bacterial poisons so lower its resistance that cirrhosis results from the effects of the latter.

(*a*) Boix described "dyspeptic cirrhosis," which is not necessarily alcoholic, but is due to the action of fatty acids, such as butyric, lactic, acetic, and valerianic; this he supports by experimental results. Clinically the frequent existence of long-continued dyspepsia is quite in accord with this hypothesis (*vide* p. 185).

(*b*) Scagliosi concludes that, though alcohol or bacterial poisons alone do not permanently damage the liver, alcohol may render persistent the lesions induced temporarily in the liver by the poisons of the specific fevers and other infections, and so induce cirrhosis. Ramond[2] adopted much the same view. He produced cirrhosis by giving alternate doses of bacterial toxins and alcohol by the mouth to animals. He found, by examination of the toxicity of the faeces in man, that alcohol does not necessarily increase the poisonous bodies in the alimentary canal; he was therefore not inclined to support the first view. He believes that alcohol acts as a cellular poison on the liver cells and thus inhibits their special function of destroying poisons; these poisons are then free to induce cirrhosis. There is not much to choose between these two views; and it is neither necessary nor possible to adopt one exclusively. It is, indeed, not improbable that both of them may be true.

To conclude: with regard to the rôle of alcohol in the production of cirrhosis, it may safely be held that alcoholism is frequently an antecedent condition, but that *per se* alcohol has no specific action on the liver except fatty degeneration. It gives rise to cirrhosis in a secondary manner, either by leading to the production of sclerogenic poisons or by enabling such poisons to have full sway on the liver. The importance of alcoholism has been rather exaggerated, and it is not sufficiently recognised that other factors may cause cirrhosis, and that a congenital or acquired want of resistance on the part of the liver itself is, though hard to estimate, probably of great importance.

(2) *Cirrhosis due to Ingested Poisons other than Alcohol.*—There are numerous cases of undoubtedly non-alcoholic cirrhosis, especially in children. In some of these there has been a definite history of high living, and occasionally two or even more members of a family have early in life become affected with hepatic cirrhosis. Cases are on record in which fish pickled in vinegar had been largely taken; and spices, curries, and highly flavoured food have long been thought to account for some cases of cirrhosis. But here it at once becomes evident that it is difficult to draw a hard-and-fast line between poisons which are ingested and

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[1] Viola. *Arch. gén. de méd.*, 1898, i, 1, 164, 318.

[2] Ramond. *Presse méd.*, 1897, p. 178.

give rise to cirrhosis by virtue of their own inherent irritating properties, and those factors which set up gastro-intestinal catarrh and by the manufacture of fermentation products inside the alimentary canal lead to what has been termed dyspeptic cirrhosis. Experimentally, it is true, Boix[1] produced some cirrhosis in rabbits by feeding them on acetic, butyric, valerianic, and lactic acids, but these acids are, on the whole, more likely to be produced by fermentation in the human alimentary canal than to be swallowed as such in food. A good example of cirrhosis due to poisonous food is reported by Segers[2] among the Fuegians, who eat large quantities of mussels daily. At a certain stage of their development the mussels are toxic from the presence of mytilotoxin. As a result of this poison the livers of the Fuegians become enlarged and subsequently cirrhotic and small.

In rare instances cirrhosis has been found to be associated with the presence in the liver, or possibly to be caused by the irritation, of particles of carbon absorbed from the alimentary canal. This form of cirrhosis—cirrhosis anthracotica—has been seen in connexion with pulmonary anthracosis, and Adami[3] refers to an analogous form associated with stonemason's lung—silicosis. Lancereaux[4] says it occurs in workers in copper and in coal-miners, and that the intestinal walls are pigmented from the presence of the carbon, this shewing the route taken in its absorption. The lymphatic glands in the abdomen are also crowded with particles of carbon. Welch[5] also described a case (*vide* p. 302).

Arsenic appears to be capable of setting up cirrhosis of the liver. In the epidemic of arsenical peripheral neuritis due to poisoned beer in the north of England in 1900–1901 an unusual number of cases of cirrhosis of the liver and ascites were seen (Reynolds,[6] Sturrock[7]). Hutchinson[8] and Hamburger[9] reported ascites in patients who had long been addicted to the medicinal use of arsenic; but in both these instances recovery followed suspension of the drug, so that the existence of cirrhosis was not proved. It has even been suggested that Banti's disease, or the terminal cirrhosis which supervenes in some cases of chronic splenic anaemia, is due to the arsenic given medicinally (W. Broadbent[10]). Experimentally cirrhosis has been produced by chronic arsenical and by chronic aluminium poisoning, but cirrhosis in man has not been traced to the latter metal.

In a case of argyria, in which nitrate of silver had been taken for four months for epilepsy, Frommann[11] found early hepatic cirrhosis with deposit of silver.

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[1] Boix. *Arch. gén. de méd.*, Paris, 1899, clxxxiv, 210.

[2] Segers. *Sem. méd.*, 1891, xi, 448.

[3] Adami. *Sajous' Annual*, 1898, ii, 313.

[4] Lancereaux. *Traité des maladies du foie et du pancréas*, p. 340, 1899.

[5] Welch. *Johns Hopkins Hosp. Bull.*, 1891, ii, 32.

[6] Reynolds, E. S. *Med.-Chir. Trans.*, 1901, lxxxiv, 425.

[7] Sturrock. *Brit. Med. Journ.*, 1900, ii, 1815.

[8] Hutchinson, J. *Arch. Clin. Surg.*, 1895, vi, 389.

[9] Hamburger. *Johns Hopkins Hosp. Bull.*, 1900, xi, 87.

[10] Broadbent, W. *Brit. Med. Journ.*, 1903, i, 1140.

[11] Frommann. *Virchows Arch.*, 1859, xvii, 135.

As a result of the admixture with wheat of the seeds or threshed portions of the weeds SENECIO ilicifolius and Burchelli cirrhosis in man has been described in South Africa (Willmott and Robertson).

Willmott and Robertson. *Lancet*, 1920, ii, 848.

Silica. Gye and Purdy produced Cirrhosis in rabbits by repeated intravenous injections of colloidal silica. Chronic poisoning by silica is not established to have this effect in man, but beer contains silica

Gye, W. E. and Purdy, W. J. Journ. Exper. Path., London, 1924, V, 238

Lead.—Experimentally some slight degree of hepatic fibrosis has resulted from feeding animals with lead (Lafitte[1]), but clinically lead has no claim to be considered a cause of cirrhosis; Lavrand,[2] indeed, states that the liver is generally unaffected in chronic plumbism. There is, moreover, some doubt as to the mechanism by which lead would produce cirrhosis; perhaps the more probable view is that it leads to arteriosclerosis of the branches of the hepatic artery, and so to slight dystrophic fibrosis.

Chloroform.—Experimentally chloroform usually produces fatty change, but Opie[3] found that when given interruptedly over a period of sixty days cirrhosis resulted. When chloroform was given by the mouth, and bacteria injected into the blood, cirrhosis was readily produced.

Naphthol.—By injecting small quantities of naphthol into the portal vein Bouchard[4] produced some fibrosis in the liver.

Bacterial Toxins.—Experimentally Charrin[5] injected boiled cultures of the *Bacillus pyocyaneus* into the portal vein, and produced extensive small-celled infiltration in the portal canals. Krawkow[6] obtained cirrhosis in birds in similar experiments when prolonged, but Ramond failed to induce cirrhosis in animals by the administration of toxins by the mouth alone, though he obtained positive results when alcohol was given as well.

Krawkow, by introducing cultures of *Staphylococcus pyogenes aureus* and of other micro-organisms into the alimentary canal produced experimental cirrhosis. The micro-organisms may have merely manufactured a poison, which passed into the portal vein and so to the liver, and set up a purely toxic cirrhosis, but, on the other hand, the micro-organisms may themselves have reached the liver either by the portal vein or by ascending the bile-duct. These points will be referred to later.

(*b*) CIRRHOSIS DUE TO THE ACTION OF POISONS MANUFACTURED IN THE INTESTINE.—This hypothesis has already been referred to (*vide* p. 183), in order to explain cirrhosis supervening in alcoholic subjects, where it was suggested that alcohol sets up gastro-enteritis, and thus leads to the formation of poisons which are the direct causes of cirrhosis. Boix[7] and Hanot[8] have described non-alcoholic cirrhosis due to dyspepsia. The liver is first enlarged and tender, and later cirrhosis and jaundice may supervene. The cirrhosis ascribed to gout may be of this nature, while the non-alcoholic cirrhosis in Mohammedans and the natives of India and Egypt, a form long ago referred by Budd[9] to the action of spices, curries, and other stimulating articles of food, such as ginger, may be explained as depending on the irritating effects of acetic, butyric, lactic, or other acids manufactured as the result of intestinal fermentation. Cirrhosis of this kind is sometimes described as due to auto-intoxication or as autochthonous, and in a posthumous article by Hanot[1] was called "Budd's cirrhosis." D'Amato[2] gave butyric acid daily for six months to animals and found parenchymatous, but no interstitial, changes in the liver. By the administration of paracresol, phenol, and indol, which are produced by the colon group, Metchnikoff[3] produced early cirrhosis in animals. In order to meet the obvious objection that dyspepsia is comparatively seldom followed by cirrhosis, Boix argues that the resistance of the liver is an important factor, and that where this is congenitally feeble continued dyspepsia will lead to cirrhosis. This idiosyncrasy or predisposition is included under the French term "arthritism."

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[1] Lafitte. *Thèse de Paris*, 1892.

[2] Lavrand. *Le Néphrite des saturnines*, p. 12, 1899.

[3] Opie. *Journ. Exper. Med.*, N.Y., 1910, xii, 367.

[4] Bouchard. *Thérapeutique des maladies infectieuses*, p. 313, 1889.

[5] Charrin. *Arch. de physiol. norm. et path.*, 1893, xxv, 554.

[6] Krawkow. *Arch. de méd. expér. et d'anat. path.*, 1896, viii, 268.

[7] Boix. "La Foie des dyspeptiques," *Thèse de Paris*, 1894; *Arch. gén. de méd.*, 1899, clxxxiv, 210.

[8] Hanot et Boix. *Congrès de méd.*, Rome, 1894.

[9] Budd. *Diseases of the Liver*, p. 151, 1857.

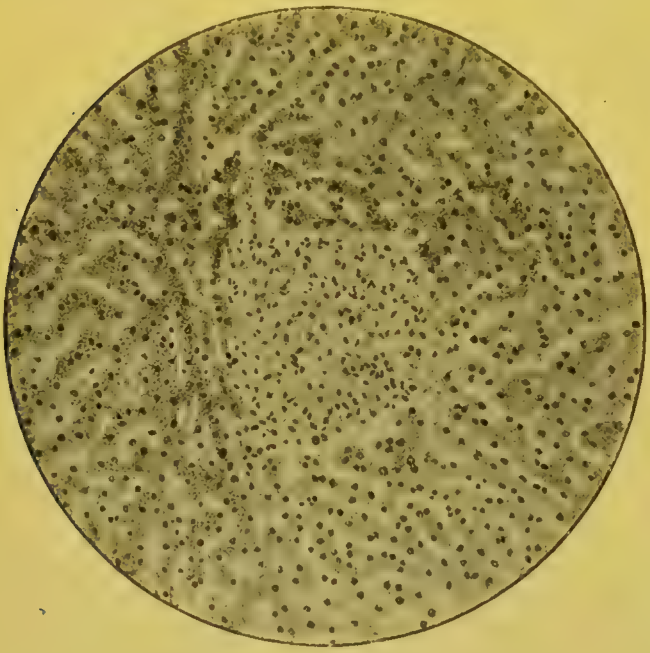

FIG. 25.—Focal necrosis in enteric fever.

In long-standing mitral disease hepatic fibrosis may be due to several causes. A certain degree of fibrous replacement may result from atrophy of the liver cells; and gastro-intestinal catarrh may give rise to the formation of irritating bodies which, when absorbed, find the liver in a condition of diminished resistance and are thus able to set up changes. The fibrosis secondary to chronic venous engorgement, however produced, is slight and of no clinical importance (*vide* p. 88).

Among Brahmins and other high-class Hindus who, as a rule, never touch alcohol, cirrhosis is frequent (Young[4]). Out of 163 cases of cirrhosis in natives of India, 138 had never touched alcohol (Sutherland[5]). Ginger-eating is prevalent with them and they are addicted to cardamoms, red pepper, and other hot spices. The cirrhosis may well be secondary to dyspeptic changes thus induced. In Egypt cirrhosis is found among Mohammedans, who take no alcohol, and may possibly be due to ankylostomiasis and the production of toxins in the intestine.

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[1] Hanot. "La Cirrhose de Budd," *Arch. gén. de méd.*, 1899, clxxxiii, 3.

[2] d'Amato. *Giorn. internaz. d. sc. med.*, Napoli, 1907, N.S., xxix, 337.

[3] Metchnikoff. *Ann. de l'inst. Pasteur*, Paris, 1910, xxiv, 755.

[4] Young, L. T. *The Carlsbad Treatment*, p. 129, 2nd ed., 1899 ("Ginger Liver").

[5] Sutherland, D. W. *Ind. Med. Gaz.*, 1905, xl, 120.

and JOANNOVICS also failed
to obtain a positive result

JOANNOVICS. *Arch. int. Pharmacodyn.*, 1905, XV, 241

In enteric fever[1] necrotic areas of small size, focal necroses, "lymphomata," or "lymphoid nodules," as they have been variously called, are often met with. The liver cells first degenerate and subsequently a certain amount of small-round-celled infiltration surrounds the necrotic areas. It is conceivable, therefore, that under favourable conditions a sporadic multiple fibrosis may result throughout the liver; usually these lesions are recovered from. Some observers have regarded enteric fever as a starting-point for cirrhosis. It is possible that if alcohol is taken and the resistance of the liver thus further diminished, this change may be rendered permanent. From Reed's and M'Crae and Klotz's[2] observations it appears probable that these focal necroses are due to toxins conveyed to the liver and possibly to thrombosis, and that these areas do not contain typhoid bacilli. Hanot found similar nodules in the liver in tuberculous enteritis. According to Mallory,[3] two distinct lesions have been described as lymphoid nodules: (1) a focus of lymphoid and plasma-cells confined to the connective tissue around the portal vessels and independent of the liver cells; (2) focal necrosis of cells in hepatic lobules due to blocking of capillaries by proliferated and phagocytic cells.

Pearce[4] has shewn experimentally that focal necroses can be directly caused by hyaline thrombosis (fused red blood-corpuscles) in the vessels, produced by intravenous injection of haemagglutinative serum, and that as the result of reparative processes around these focal necroses well-marked cirrhosis occurs in dogs.

Cirrhosis has been regarded as a late result of cholera; and it seems reasonable to believe that the slightly increased fibrosis around the portal spaces sometimes seen in rickets is due to gastro-enteritis. On the other hand, prolonged diarrhoea and gastro-enteritis in children do not play any part in the etiology of cirrhosis. It is conceivable that toxins absorbed in the course of appendicitis[5] may initiate cirrhotic changes in the liver.

By intraperitoneal injection of a micro-organism of the colon group Weaver[6] obtained cirrhosis in guinea-pigs, but the micro-organism was not found in the liver, hence the change in the liver may be regarded as due to the toxins absorbed by the portal vein.

(c) UMBILICAL VEIN CIRRHOSIS.—In *congenital syphilis*, the blood containing the pathogenetic spirochaete arrives by the umbilical vein and thus passes into the branches of the portal vein in the liver. The resulting lesion is a diffuse unicellular cirrhosis. In exceptional instances a similar change is found in acquired syphilis; but otherwise syphilis does not directly produce hepatic cirrhosis, though it may lead to local fibrosis, gummas, and cicatrices. Though this is undoubtedly true, it appears that unicellular cirrhosis, which is a secondary lesion of syphilis and therefore curable, often leaves behind a deficient resistance on the part of the liver and thus disposes it to become cirrhotic in the ordinary way. I have seen several cases of multilobular cirrhosis in the subjects of congenital syphilis. In other words, congenital syphilis so prepares the soil that ordinary cirrhosis may supervene. In these conditions the cirrhosis may be regarded as a parasyphilitic lesion.

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[1] Friedreich, *Virchows Arch.*, 1857, xii, 53; Reed, *Johns Hopkins Hosp. Rep.*, v, 379; Handford, *Trans. Path. Soc.*, 1889, xl, 129; Hanot, *Compt. rend. Soc. Biol.*, 1893, xlv, 856; l.c Siredey, *Rev. de méd.*, 1886, vi, 465; Legrey, *Thèse de Paris*, 1892.

[2] M'Crae and Klotz. *Journ. Path. and Bacteriol.*, Cambridge, 1908, xii, 279.

[3] Mallory. *Journ. Exper. Med.*, N.Y., 1898, iii, 622.

[4] Pearce, R. M. *Ibid.*, 1906, viii, 64.

[5] Tuffier et Mauté. *Presse méd.*, 1904, p. 408.

[6] Weaver. *Johns Hopkins Hosp. Rep.*, 1900, ix, 543.

Other poisons may travel from the mother to the fetus by the umbilical vein and give rise to changes in the liver. Our knowledge of umbilical vein cirrhosis is extremely meagre, but it is possible that the disease known as congenital obliteration of the bile-ducts may start in the liver and be due to toxins conveyed by the umbilical vein, and that the subsequent obliteration of the ducts is a later change due to a descending cholangitis (*vide* p. 651).

(*d*) POISONS MANUFACTURED IN THE SPLEEN.—In haemic infections micro-organisms tend to accumulate in the spleen, and, if not rapidly destroyed, produce toxins which travel to the liver and may there produce changes analogous to cirrhosis. Chauffard[1] has argued that cirrhosis is in some cases secondary to morbid processes originating, or at any rate most marked, in the spleen. Thus, in malaria the spleen is greatly enlarged and cirrhosis of the liver in malarial subjects may in part be due to poisons manufactured in the spleen. In chronic splenic anaemia a terminal cirrhosis of the liver with ascites ~~jaundice~~ sometimes occurs (Banti[2]); this has been termed Banti's disease. The conception of Banti's disease has been much criticised; Gilbert and Lereboullet[3] insist that the splenic enlargement is due to passive engorgement produced by latent hepatic lesions.

As will be seen in the description of hypertrophic biliary cirrhosis, the typical unilobular cirrhosis is very frequently obscured by the addition of multilobular cirrhosis. This latter change may be explained as due to the infective agent, which primarily reached the liver by the hepatic artery and sets up unilobular cirrhosis, settling down in the spleen and producing toxins which, when conveyed by the splenic vein to the liver, induce multilobular cirrhosis. In support of the hypothesis that poisons are manufactured in the spleen and conveyed to the liver is the fact that endophlebitis of the splenic vein has been described in malaria by Kelsch and Kiener,[4] in enteric fever by Bezançon[5] and in splenic anaemia (Oettinger and Fiessinger[6]).

The portal vein may indeed be regarded as having two main sources in adult life—(*a*) the gastro-intestinal veins, by which poisons commonly travel to the liver, and (*b*) the splenic vein. In fetal life the umbilical vein is a third tributary and may also convey poisons to the liver.

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[1] Chauffard. *Semaine méd.*, Par., 1899, xix, 177.

[2] Banti, G. Semaine méd., Par., ~~Ibid.~~, 1894, xiv, 318.

[3] Gilbert et Lereboullet. *Rev. de méd.*, Par., 1904, xxiv, 893.

[4] Kelsch et Kiener. *Traité des maladies des pays chaudes*, p. 405, 1889.

[5] Bezançon, F. *Thèse de Paris*, 1895.

[6] Oettinger et Fiessinger. *Rev. de méd.*, Par., 1907, xxvii, 1109.

In Egyptian splenomegaly Cirrhosis of the liver eventually results (Richards and Day)

(Vide pp. 6[illegible], 366)

In the Gaucher or familiar form of splenomegaly hepatic Cirrhosis has also been described (Mandlebaum).

Richards and Day. *Trans. Soc. Trop. Med.* and Hyg., 1912, v, 333.

Mandlebaum. *Journ. exper. Med.*, 1912, xvi, 797.

Sérégé,[1] from injection experiments with methylene-blue, concludes that the blood from the spleen goes exclusively to the left lobe of the liver. If this is true, cirrhosis due to poisons produced in the spleen should be confined to, or chiefly in, the left lobe of the liver. ~~I am not aware tha~~t there is ~~any~~ evidence ∧/Very lit in support of this.

At present the existence of hepatic cirrhosis as a sole result of poisons manufactured in the spleen requires further investigation, but it is an attractive idea and is compatible with the fact that in one variety of hypertrophic biliary cirrhosis, the so-called metasplenomegalic, the spleen is enlarged before there is any manifest change in the liver.

II. MICRO-ORGANISMS CONVEYED TO THE LIVER BY THE PORTAL VEIN

Cirrhosis thus produced would be described as infective in contradistinction to toxic cirrhosis, which is due to the action of poisons without the presence of microbes in the liver. This view is highly probable, but cannot be accepted at present. Hamilton[2] suggested that micro-organisms passing into the mesenteric veins undergo bacteriolysis in the portal blood, and that their toxins are carried to the liver and set up cirrhosis, bacterial cirrhosis. Adami,[3] in the Pictou cattle disease, found unicellular cirrhosis accompanied by swelling of the peri-portal and retroperitoneal lymphatic glands, and oedema of parts of the intestine. These lesions were associated with a minute bacillus belonging to the colon group. Adami has found the colon bacillus almost constantly in human livers; when the livers are healthy, the bacilli appear to have been killed by the liver cells. In progressive cirrhosis there are, in addition to dead bacilli, some areas where they appear active. This suggests the possibility that virulent colon bacilli may under conditions of diminished resistance of the liver cells, such as may be induced by alcohol, lead to cirrhosis. Adami[4] considered that a primary inflammation of the alimentary canal favoured invasion of the liver by the bacilli which set up cirrhotic changes in that organ.

It is, a priori, extremely probable that an acute hepatitis due to the brunt of a haemic infection falling on the liver would, if not fatal, leave behind it hepatic cirrhosis. Cases in human beings occur in which a febrile condition, accompanied by enlargement and tenderness of the spleen and liver, ushers in and precedes an illness which eventually is seen to have for its chief anatomical lesion a cirrhotic liver. Oertel[5] points out that if in acute hepatitis the lymphatics become obstructed from lymphangitis and perilymphangitis, resolution is prevented and cirrhosis results.

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[1] Sérégé, H. *Journ. de méd. de Bordèaux*, 1902, xxxii, 357.

[2] Hamilton. "The Alimentary Canal as a Source of Contagion," p. 37, *Studies in Pathology*, Aberdeen Quater-Centenary, 1906.

[3] Adami. *Brit. Med. Journ.*, 1898, ii, 1215.

[4] *Idem.* *Report of Minister of Agriculture for the Dominion of Canada for the Year 1901*, p. 135.

[5] Oertel. *Arch. Int. Med.*, Chicago, 1908, i, 385.

In a boy, aged sixteen years, death occurred fourteen days after the onset of measles. Roger and Conte[1] found that an acute localised enteritis in the ileum had given rise to hepatitis, inflammation between the liver and diaphragm, an acute rapid putrid effusion into the right pleura, and, finally, gangrene of the lung. The bacillus resembled the *B. coli*. The liver shewed masses of round-celled infiltration in the portal spaces, degeneration of the liver cells, cholangitis, and endarteritis. The process was very acute, but it makes it easy to understand that a slighter degree of the same process, if recovered from, might be the starting-point of cirrhosis.

Some fibrosis is seen in prolonged cases of acute yellow atrophy. By intraperitoneal injections of cultures of a bacillus belonging to the pseudo-diphtheria group Hektoen[2] produced cirrhosis of the liver in animals. The bacilli were found in the capillaries of the liver.

By introducing cultures of micro-organisms into the intestines of birds Krawkow sometimes produced cirrhosis ; but the result may have been merely due to their toxins, for there is no evidence that the micro-organisms reached the liver.

It is possible that in exceptional instances tubercle bacilli carried from the intestines to the liver may set up some cirrhosis. Hanot and Gilbert's[3] experiments shew that avian tubercle bacilli may induce cirrhosis in guinea-pigs, and they believe that this sclerogenic result depends either on relatively high resistance on the part of the liver or on a slight degree of virulence on the part of the bacilli. As a rule, of course, tubercle bacilli give rise either to tubercles or to degeneration in the liver cells (*vide* p. 345). The ova of bilharzia may cause cirrhosis;[4] ∧ Kartulis[5] states that the liver is usually enlarged and that symptoms of cirrhosis are absent, but according to Phillips[6] it may cause ascites. Cirrhosis may also be set up by *Schistosomum japonicum* (Catto[7]). Michelazzi[8] described cirrhosis due to ankylostomiasis. Day[9] considers that in native Egyptians cirrhosis is probably due to some specific protozoan infection.

III. CIRRHOSIS DUE TO POISONS CONVEYED TO THE LIVER BY THE HEPATIC ARTERY

This cirrhosis may be spoken of as toxic and due to the action of poisons on the liver. It has been suggested that cirrhosis may be set up by the specific fevers, typhoid fever, scarlet fever, measles, small-pox,

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[1] Roger et Conte. *Presse méd.*, 1897.

[2] Hektoen. *Journ. Path. and Bacteriol.*, Edin. and London, 1901, vii, 214.

l.c [3] Hanot et Gilbert. *Compt. rend. Soc. Biol.*, Paris, 1892, xliv, 72.

[4] Symmers. *Journ. Path. and Bacteriol.*, 1904, ix, 237.

[5] Kartulis. *Sem. méd.*, 1894, xiv, 415.

[6] Phillips. *Rec. Egyptian Government School of Medicine*, 1904, ii, 49.

[7] Catto. *Brit. Med. Journ.*, 1905, i, 11.

[8] Michelazzi. *Giorn. internat. d. sc. med.*, Naples, 1907, xxix, 241.

[9] Day. *Lancet*, Lond., 1909, i, 258. See also Ferguson and Day. *Ann. Trop. Med.*, Liverpool, 1909, iii, 379.

^] according to Ferguson this occurs in about 3 per cent of the cases of bilharziasis & is characterized by great hyperplasia of the interlobular fibrous tissue, which contains ova with lateral spines. Bilharzial cirrhosis is also common in South Africa (PIRIE).

Whitmore

[illegible]
[illegible]

Ferguson. Glasgow Med. Journ., 1913, LXXIX, 21
PIRIE. Med. Journ. South Africa, 1921, XVII, 87.
Whitmore. Arch. int. Med., Chicago, 1908, i, 430.

[illegible]

and pneumonia. Theoretically, the toxins of these diseases may, when absorbed into the general circulation, set up focal necrosis of the liver cells, and under certain conditions fibrosis might develop around these areas of necrosis.

Focal necrosis may occur in typhoid, scarlet fever, measles, small-pox, diphtheria, malaria, and other acute infections. In typhoid fever the poison might reach the liver by the hepatic artery and give rise to focal necrosis. Klein[1] and Crooke[2] described acute interstitial hepatitis in scarlet fever, and more recently Pearce[3] has met with focal necroses of the liver cells; this is analogous to scarlatinal nephritis, but it probably is a transient condition and clinically there is little relation between scarlet fever and hepatic cirrhosis. Focal necroses in measles have been described and figured by Freeman.[4] Small-cell‸accumulations, like those ‸|- ED produced in typhoid fever, have been described in the liver in variola by Roger and Weil.[5] Arnaud,[6] who describes in addition small-celled infiltration near the portal spaces, suggests that cirrhosis may subsequently follow.

In diphtheria focal necroses occur round the intralobular vein (Councilman, Mallory, and Pearce[7]), with some proliferation of the endothelium. These changes are due to the action of poisons and not to the presence of diphtheria bacilli in the liver. Weaver[8] injected a micro-organism allied to the colon group into the abdominal wall of guinea-pigs and produced local abscesses in which the bacilli were found, none being present in the liver, which shewed focal necroses and cirrhosis. It is reasonable to believe that as the result of other local infections focal necroses in the liver may be produced, and that under favourable conditions some sporadic fibrosis might develop around these foci. The question whether absorption from tuberculous pulmonary vomicae which are secondarily infected with streptococci can induce cirrhosis is referred to elsewhere (p. 347).

The conditions favouring the development of permanent cirrhosis after infections are congenital susceptibility, want of resistance on the part of the liver, and the presence of other factors, such as alcoholism.

Experimentally, a certain amount of hepatic fibrosis has followed prolonged poisoning with vegetable, mineral, and bacterial poisons. The intoxication must be induced gradually and continued for a considerable period. If the poison is employed in too large amounts, the results are those of acute poisoning, viz. degeneration and necrosis of the liver cells, focal or diffused, as in phosphorus poisoning. In the same way, the injection of bacterial poisons into the circulation, when carried on rapidly, gives rise to necrotic changes in the liver cells around the intralobular veins; when the process is less intense and more prolonged a certain amount of cirrhosis results. Krawkow[1] obtained positive results with sterilised cultures of *Bacillus pyocyaneus*, and Claude[2] with diphtheritic toxin. But it is probable that these effects are transitory, and do not, when uncomplicated (*vide* p. 183) by alcoholism, induce cirrhosis in man. Flexner,[3] in a careful study of chronic intoxication by ricin and abrin—vegetable alkaloids or phytalbumoses—produced a form of cirrhosis. As a result of injection of phosphorated oil into the subcutaneous tissues of rabbits, Aufrecht[4] found that the liver cells shewed degeneration, and that later some small-celled infiltration took place around the lobules. Other poisons, when injected into the circulation, such as carbonate of ammonia, indol, skatol, phenol, sulphonal, have also led to a slight degree of microscopic fibrosis.

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[1] Klein. *Trans. Path. Soc.*, 1877, xxviii, 439.

[2] Crooke. *Birmingham Med. Rev.*, xx, xxi.

[3] Pearce. *Boston (U.S.A.) City Hosp. Rep.*, 1899, p. 74.

[4] Freeman. *Arch. Pediat.*, 1900, xvii, 81.

[5] Roger et Weil. *Compt. rend. Soc. Biol.*, 1900, lii, 911.

[6] Arnaud. *Marseille méd.*, 1899, p. 39.

[7] Councilman, Mallory, and Pearce. *Diphtheria, a Study of 220 Fatal Cases*, Boston, U.S.A., 1901.

[8] Weaver. *Johns Hopkins Hosp. Rep.*, Balt., 1900, ix, 543.

IV. CIRRHOSIS DUE TO MICRO-ORGANISMS CONVEYED TO THE LIVER BY THE HEPATIC ARTERY

In haemic infection the liver changes are often extremely acute and give rise either to suppuration or to widespread degenerative changes allied to acute yellow atrophy. In less acute haemic infections the supporting fibrous tissues shew proliferation and accumulation of leucocytes in the portal canals and in the peripheral parts of the lobules of the liver. These changes are the same as those seen in the acute specific fevers, such as scarlet fever (*vide* p. 191). Occasionally in pyaemia and septicaemia, due to streptococci, staphylococci, etc., as in acute necrosis, infective endocarditis, puerperal septicaemia, and erysipelas,[5] considerable small-celled infiltration of the liver is met with.

In the hepatitis due to repeated malaria there is considerable damage done to the liver cells, as shewn by focal necroses (Barker[6]), and fibrous hyperplasia is sometimes seen. The importance of malaria as a cause of cirrhosis appears to be surprisingly small (*vide* p. 308). Rogers[7] describes cirrhosis due to the Leishman-Donovan parasite of kala azar.

Enteric fever has already been referred to as leading to small necrotic areas in the liver (p. 191). In ordinary enteric fever these nodules appear to be due to the action of toxins conveyed from the alimentary canal, and not to bacilli. But infection of the liver by the hepatic artery may also occur. The changes in the liver due to general haemic infections are probably usually recovered from if the patient lives, but should conditions arise which depress the resistance of the liver, such as alcoholism, cirrhosis might conceivably result. This is supported by Opie's[8]

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[1] Krawkow. *Arch. de méd. expér. et d'anat. path.*, 1896, viii, 269.

[2] Claude. *Thèse de Paris*, 1897.

[3] Flexner. *Journ. Exper. Med.*, N.Y., 1897, ii, 19.

[4] Aufrecht. *Deutsch. Arch. f. klin. Med.*, 1897, lviii, 302.

[5] Roger et Garnier. *Rev. de méd.*, 1901, xxi, 97.

[6] Barker. *Johns Hopkins Hosp. Rep.*, 1895, v, 241.

[7] Rogers. *Ann. Trop. Med. and Parasit.*, 1908, ii, 417.

[8] Opie. *Journ. Exper. Med.*, N.Y., 1910, xii, 367.

; and according to Nattan-Larrier the cirrhosis may be nodular or diffuse and around the capillaries, as in congenital syphilis, the Leishman bodies being seen in the connective tissue.

← In ~~a case of~~ the Cirrhotic ~~a~~ liver of a leper the leprosy bacillus has been found ~~in the liver~~ (Carrieu and Anglada).

Nattan-Larrier, L. Bull. Acad. de méd., Par., 1918, 3e ser., LXXIX, 402

CARRIEU et Anglada. *Arch. de méd expér. et d'anat. path.*, Par., 1913, XXV, 149

Syphilis has been considered to give rise to cancer quite distinct from those of peptic ulcer but Symmers [illegible] Judging from the frequency of a positive Wassermann reaction in peptic ulcer cases the old idea that peptic ulcer may be syphilitic. Among 1[illegible] cases of which 14 [illegible] positive Wassermann reaction (Smithie and [illegible]). Among 19 cases of peptic ulcer Olson found [illegible] to be syphilitic, among Chauffard and [illegible] 83 cases of ulcer 11 were syphilitic

√ Symmers, D. Journ. Am. Med. Assoc., Chicago 1916, LXVI, 1457

[illegible] Tratamiento y Paris 1918. [illegible]

[illegible] 1921, V, 20.

[illegible] 1924 [illegible]

[illegible]

observation that administration of chloroform accompanied by intravenous injection of *B. coli* readily sets up cirrhosis.

Experimentally there is some evidence that haemic infections may induce hepatic cirrhosis. Krawkow[1] injected cultures of various micro-organisms into the muscles of fowls and pigeons, and found that after a considerable interval hepatic cirrhosis developed. This was especially well marked when the *Bacillus pyocyaneus* and the *Staphylococcus pyogenes aureus* were employed. By subcutaneous injection of cultures of a bacillus belonging to the pseudo-diphtheria group, Hektoen[2] obtained well-marked portal cirrhosis in animals.

It must, however, be remembered that in chronic haemic infections the spleen becomes crowded with micro-organisms, and that if they multiply there, toxins may be manufactured in considerable quantities and carried by the splenic and portal veins to the liver, and there set up a cirrhosis of splenic origin (*vide* p. 188).

Conclusion.—Experiment shews that numerous poisons are capable of giving rise to hepatic changes comparable to those of cirrhosis. Often, it is true, these lesions are early, or, at the best, not well marked. But the facts are of value as indicating that cirrhosis in man may reasonably be considered to be the result of a toxic process. These poisons may be absorbed either from the alimentary canal, and then reach the liver in a comparatively concentrated form, or they may travel to the liver by the hepatic artery, and are then comparatively dilute. Ordinary cirrhosis in man is generally due to poisons travelling by the portal vein. Alcoholism is rather an antecedent condition than a true cause, and acts indirectly or in an accessory manner. The possibility that cirrhosis is definitely due to micro-organisms must be faced; from analogy it is most probable, but at present it has not been certainly established. It is also highly probable that poisons, or perhaps micro-organisms, reaching the liver by the hepatic artery, may give rise to cirrhotic changes. The mechanism of this change will be further referred to in the section on "Hypertrophic Biliary Cirrhosis." As a result of destruction of the hepatic cells and absorption of their proteins into the circulation, hepatic anti-bodies or cytolysins are produced. These destroy the liver cells and thus a vicious circle is produced (Fiessinger[3]).

Nature of the Fibrosis of Cirrhosis.—It was formerly held that cirrhosis is primarily and essentially a chronic inflammation or hyperplasia of the connective tissue in the portal areas, and that the atrophic and degenerative changes in the liver cells are either (i) entirely secondary and due to pressure exerted by the contracting fibrous tissue, or to impaired nutrition from curtailed blood supply, or (ii) unimportant and almost accidental concomitant phenomena.

Subsequently it was thought that cirrhosis is essentially a replacement fibrosis and is secondary to a primary degeneration and atrophy of the liver cells (Payne,[1] L. Beale). The changes in cirrhosis of the liver would thus be regarded as analogous to those in systemic scleroses of the spinal cord or to those in an arteriosclerotic kidney. Against this it may be urged—

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[1] Krawkow. *Arch. de méd. expér. et d'anat. path.*, 1896, viii, 268.

[2] Hektoen. *Journ. Path. and Bacteriol.*, Edin. and Lond., 1901, vii, 214.

[3] Fiessinger. *Journ. physiol. et path. gén.*, Paris, 1908, x, 671.

1. That extensive atrophy or degeneration may occur without any fibrosis resulting. Thus, in old age there may, as I have seen, be marked atrophy of the cells in the peripheral zone of the hepatic lobule, with little or no fibrosis. In extensive central necrosis of the hepatic lobules the fibroblasts do not proliferate when the liver cells alone are destroyed (Mallory [2]). In lardaceous disease and in universal fatty change there may be complete freedom from fibrosis. In reply, it may be said that in such cases there is a general debility of the whole organ, and that the fibrous tissues share in it, and are therefore unable to proliferate (Kanthack[3]). ~~MacCallum[4] has reported marked local fibrosis around an extensive area of degeneration set up during acute illness.~~

2. That, microscopically, the proliferation of the connective tissue is so exuberant, so like that of active inflammation elsewhere, and the degenerative changes in the cells so slight in comparison, that it is difficult to believe that the latter changes can be primary. In such cases there may well be two factors at work, namely, proliferation of the connective tissues and degenerative changes in the liver cells, and both may be due to the simultaneous action of the toxin. But, while bearing this explanation in mind, it must be remembered that a replacement fibrosis shews itself, not only as a passive overgrowth, but as a proliferation, for, both in degenerative "neuritis" and in granular kidney (arteriosclerotic in origin), much small-celled proliferation may sometimes be seen. Again, the absence of any extensive degeneration of the liver cells in cirrhosis must not be allowed to weigh too heavily, for the foci of degenerated cells may have disappeared. Kretz,[5] MacCallum,[6] and Milne [7] regard cirrhosis as a process of repair and as the result of successive regenerations of repeated focal lesions of the liver cells at the peripheries of the lobules. The frequent degenerations and subsequent regenerations of the hepatic cells lead to a progressive fibrosis which subsequently contracts. This view is supported by Pearce's [8] observations that cirrhosis follows focal necroses set up by hyaline thrombosis of the vessels.

3. That inasmuch as cirrhosis is, in ordinary conditions, due to the action of a poison, it is reasonable to believe that the effects will be no more limited to the hepatic cells than to the fibrous tissues, as was formerly

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[1] Payne, J. F. *Trans. Path. Soc.*, Lond., 1889, xl, 321.
[2] Mallory. *Johns Hopkins Hosp. Bull.*, Balt., 1911, xxii, 69.
[3] Kanthack. *St. Barth. Hosp. Journ.*, Lond., 1896, iv, 22.
~~[4] MacCallum. *Journ. Amer. Med. Assoc.*, 1906, xlvii, 984.~~
[5] Kretz. *Wien. klin. Wchnschr.*, 1900, xiii, 271.
[6] MacCallum. *Journ. Amer. Med. Assoc.*, 1904, xliii, 649.
[7] Milne. *Journ. Path. and Bacteriol.*, Cambridge, 1909, xiii, 127.
[8] Pearce, R. M. *Journ. Exper. Med.*, 1906, viii, 64.

supposed to be the case. In other words, that the parenchymatous and interstitial changes are both, in the first instance, due to the same cause, and therefore vary directly in intensity. If this be so, it is easy to understand that the hepatic cells, being more sensitive, will react before the more resistant connective tissue, and that degenerative changes in them will generally, for this is their usual method of reacting, precede hyperplasia of the connective tissues. There are further grounds supporting the view of a simultaneous reaction on the part of the liver cells and framework as the starting-point, or first stage at any rate, in some cases of cirrhosis. In early cirrhosis the liver cells may shew evidence of active proliferation in the nuclear mitoses, while at the same time there is fibrosis. This process might perhaps be regarded as a general hyperplasia, and many cirrhoses, such as the nodular cirrhosis sometimes seen in malaria or tuberculosis, might be spoken of as hypertrophies that failed.

But it is important to bear in mind that there are possibly, if not probably, different ways in which the fibrosis of cirrhosis is brought about, and that the causative factors may vary during the course of an individual case. Thus, while both the changes may at the outset be the result of the toxin, irritative hyperplasia in one case, degeneration in the other, subsequently one of these changes may react on the other process.

In conclusion, it is, in the present state of our knowledge, safest not to regard the cirrhotic process as exclusively due either to irritative hyperplasia of the connective tissues, the hepatic cells being quite passive, on the one hand, or to primary degenerative or atrophic changes in the hepatic cells with a resulting replacement fibrosis on the other, but to steer a middle course, and to take the view that the irritating poisons leading to cirrhosis affect both elements in different ways, and that the resulting changes in one tissue may further initiate fresh changes in the other tissue, or modify those already existing.

Morbid Anatomy.—*Size and Weight.*—The size of the liver in ordinary cirrhosis varies very greatly; it may be smaller than natural, and is then often spoken of as "atrophic," and in extreme instances may not weigh more than 30 ounces; or, on the other hand, it may be very large and weigh as much as 150 or 200 ounces. A cirrhotic liver which is actually smaller than natural often weighs as much as a healthy liver or even more, its specific gravity being much increased. There are transitions between a small and a large multilobular cirrhotic liver, but they all belong to the same category of disease. It is undesirable to speak of the small ones as "atrophic" and the large ones as "hypertrophic" cirrhosis, since the practice of calling a large multilobular cirrhotic liver "hypertrophic" has caused much confusion with hypertrophic biliary cirrhosis.

The large size of some cirrhotic livers may be due to considerable fatty change in the liver cells. It has been assumed, I believe incorrectly, that increased size and fatty change in cirrhosis are specially connected with over-indulgence in malt liquors rather than in spirits.

These points were considered in the course of an analysis of 114 cases of cirrhosis undertaken by Fenton and myself.[1] Taking an equal number of alcoholic cirrhotic livers from cases in which malt liquors on the one hand and spirits on the other had been chiefly drunk, there was found to be very little difference between the average weights of the livers in the two series. The number of cases available was very small, since in most of the 114 cases in which the form of stimulant taken is mentioned, both beer and spirits were taken. In 10 beer livers the average was 69 ounces, and in 10 spirit livers 67 ounces. The spirit livers appeared to be more frequently fatty. Analysis of Foxwell's cases points to the same conclusion.[2] A series of microscopic examinations shewed that fatty change occurred in an equal proportion in large and in small cirrhotic livers, and that half the large livers examined did not shew fatty change.

In cirrhosis complicating pulmonary tuberculosis the liver is usually somewhat enlarged from fatty change. As will be shewn later (p. 197), the liver is larger in cases of cirrhosis in which the disease is latent, and death occurs from some independent cause, than in those cases that die directly from cirrhosis. The enlargement is chiefly due to compensatory hyperplasia of the liver cells, but there may be considerable fatty change in the hepatic cells, as in the form specially described by Hanot and Gilbert[3] as "hypertrophic alcoholic cirrhosis." In other instances the large size of the liver is due to the fibrosis having a smaller mesh and approaching a unilobular type or shewing that arrangement in parts. Enlargement of a cirrhotic liver is often temporary and due to congestion, either active, when the cirrhotic process is progressing, or passive and due to backward pressure from dilatation of the heart.

In cases dying from the effects of cirrhosis the liver is larger in younger subjects than in those of more advanced years. There are some grounds for thinking that in patients dying from cirrhosis in whom no alcoholic history is forthcoming, the liver is smaller than in the more familiar type of alcoholic patients dying from cirrhosis.

In 6 cases in which cirrhosis was fatal and in which there was no evidence of alcoholic excess the average weight of the liver was 41 ounces, whereas in 36 fatal cases of alcoholic cirrhosis the average weight was 67·7 ounces.

In latent cirrhosis the liver is a little larger in patients addicted to alcoholic excess than in the non-alcoholic, but the difference is very small as compared with that mentioned above in patients dying from the effects of cirrhosis.

In 29 cases in which death was due to some other cause and evidence of alcoholic excess was forthcoming, the average weight of the cirrhotic liver was 62·2 ounces, whilst in 26 cases in which alcoholic cirrhosis was latent the average weight of the liver was 67·9 ounces.

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[1] Rolleston and Fenton, W. J. *Birmingham Med. Rev.*, 1896, xl, 198.

[2] Foxwell, A. *Ibid.*, 1896, xxxix, 221.

[3] Hanot et Gilbert. *Bull. et mém. Soc. méd. des hôp. de Paris*, 1890, 3. s., vii, 492.

Sex does not exert any very special influence on the weight of the cirrhotic liver.

In 116 men the average weight of the cirrhotic liver was 61·5 ounces, or 8½ ounces in excess of 53 ounces, the normal weight, while in 41 females the average weight was 55·5 ounces, or 10 ounces in excess of the normal weight, 45 ounces.

The large cirrhotic livers are less knobby than the smaller examples which especially merit the title "hobnailed." When both the liver and the spleen are much enlarged, the left lobe of the liver may touch or overlap the spleen.

The weight of the liver in portal cirrhosis may be diminished, normal, or increased, but it is less often below than above or of the normal weight.

In 142 cases tabulated by Price[1] the liver weighed less than normal in 27 per cent.

My own statistics shew a very considerable increase in the weight of the cirrhotic liver, but in 100 cases collected by Hawkins[2] the average weight was 52 ounces, and in 93 tabulated by Kelynack[3] 53 ounces, which is very close to Reid's average weight for the normal liver (53 ounces for males, 45 ounces for females). The average weight of the liver in cases dying from the effects of cirrhosis appears to be less than that of patients with latent cirrhosis who die from independent causes.

In 155 consecutive necropsies on patients with cirrhotic livers at St. George's Hospital the average weight was 63·6 ounces. Of these, 75 died from the effects of cirrhosis, the average weight of the liver being 60 ounces ; the average weight of the liver in the remaining 80 patients who had cirrhotic livers but died from other causes was 67 ounces. In 11 of the 142 cases tabulated by Price death was due to accident, and the average weight of these cirrhotic livers was 76 ounces.

In latent cirrhosis the weight of the liver diminishes as age advances ; this is probably a senile atrophic change. In this connexion it is interesting to note that in cases of latent cirrhosis in fatal granular kidney, which is perhaps a premature senile change, the weight of the liver is not increased.

In 8 cases of combined hepatic and renal cirrhosis examined at St. George's Hospital the average weight of the liver was 56·5 ounces, the average age of the patients being 52·25 years, or 3·5 years above the average age at death in cirrhosis. Pitt's[4] figures pointed to the same conclusion, but Price's figures, also from Guy's, tended to shew that granular kidney was associated more often with a large than with a small cirrhotic liver.

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[1] Price. *Guy's Hosp. Rep.*, 1884, 3. s., xxvii, 295.
[2] Hawkins. *System of Medicine*, 1908, iv, Part i, 173.
[3] Kelynack. *Birmingham Med. Rev.*, 1897, xli, 86.
[4] Pitt, G. N. *Trans. Path. Soc.*, Lond., 1889, xl, 348.

In cases dying from cirrhosis the weight of the liver becomes less as age advances; this, again, may be partly due to the advance of years.

Morbid Appearances in the Liver.—The peritoneal surface of the liver is opaque; this may be due to chronic peritonitis, which is sometimes (*vide* p. 223) associated with cirrhosis. Not uncommonly there is more thickening of the peritoneum over the liver than elsewhere. The opacity of the capsule is, however, largely due to subcapsular fibrosis and atrophy of the liver cells, which, though it gives somewhat the same naked-eye appearance, is essentially different from genuine perihepatitis. It is probable that chronic perihepatitis is often described when subcapsular fibrosis is the actual condition present. This opacity is not uniform; it is more marked between the projections or hobnails, where there is underlying fibrous tissue.

General perihepatitis occurred in 15 out of 53 cases of cirrhosis recorded by Cheadle,[1] or 28 per cent, and in 13 out of 78 necropsies tabulated by Sears and Lord,[2] or 16·6 per cent.

There may be adhesions between the liver and adjacent parts, especially the diaphragm. These adhesions may be dense, but are often comparatively delicate. They may be remarkably vascular. The surface of the organ is irregular; the projections vary in size from a pea to that of a pigeon's egg. When they are small, the surface of the organ somewhat resembles that of a granular kidney, and the term "granular liver" is applicable. When, as more rarely occurs, the hobnails are large, the organ may look as if it were occupied by numerous secondary growths, especially when the projections shew marked fatty change and appear white, but the hobnails never shew the umbilication usually present in secondary carcinoma. When the projections are exceptionally large, the condition is sometimes spoken of as nodular cirrhosis or cirrhosis with multiple adenoma (*vide* p. 459).

Occasionally in an otherwise uniformly cirrhotic liver there is one large prominent hobnail; this has been described as solitary adenoma with cirrhosis. It is probable that this condition is of the same nature as multiple adenoma in cirrhosis, but differs in there being one area only of marked hyperplasia. Caminiti[3] collected four examples of solitary adenoma with cirrhosis.

The hobnails are tawny yellow or brown in colour, being often stained by bile; the peritoneum over them sometimes shews dilated vessels. During life the liver looked uniformly red in the laparotomies on cases of cirrhosis I have seen. The capsule, which is not much thickened as a rule, is more opaque in the depressions between the nodules. Usually the liver is uniformly affected, especially when it is enlarged and the nodules are small, but the change may be irregular and the left lobe is

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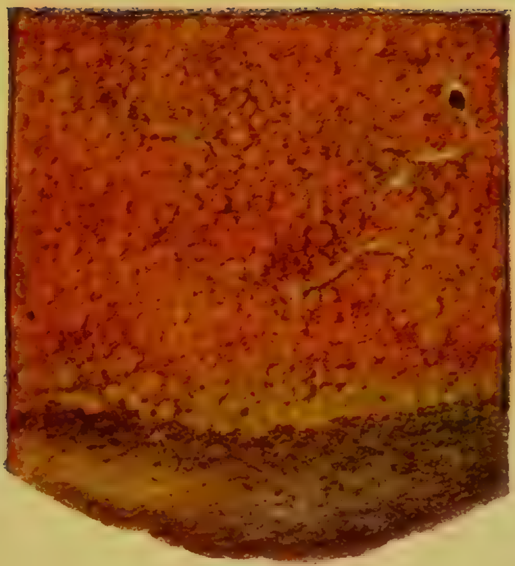
[1] Cheadle, W. B. *Lancet*, 1900, i.

[2] Sears and Lord. *Boston Med. and Surg. Journ.*, 1902, cxlvii, 285.

[3] Caminiti. *Arch. f. klin. Chir.*, 1903, lxix, 630.

PLATE II.



Portion of a Finely Granular Cirrhotic Liver Shewing the Surface and the Section.
Dr. E. A. Wilson.

often in a more advanced condition and may be very small. It is possible that the resistance of the left lobe is less than that of the right, for it is not infrequently more affected in acute yellow atrophy than the right. Sometimes, on the other hand, one of the smaller lobes, such as the Spigelian or caudate, may be enlarged out of proportion to the others, even when the organ as a whole is little, if at all, bigger than normal. The process is one of compensatory hypertrophy. The enlarged Spigelian lobe may be so blended with the right lobe as to form a complete tunnel for the inferior vena cava (Fig. 27). During the course of a Talma-Morison operation on a patient of mine the enlarged Spigelian lobe was regarded as evidence of malignant disease.

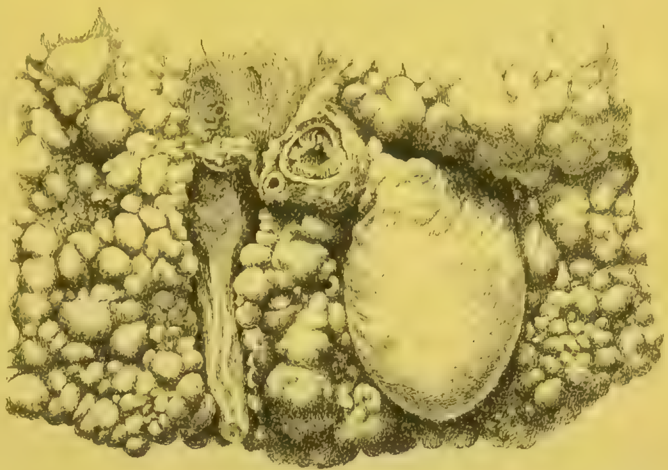


FIG. 26.—Under surface of cirrhotic liver shewing hobnails and thrombosis of the portal vein. (Drawn by Dr. E. A. Wilson.)

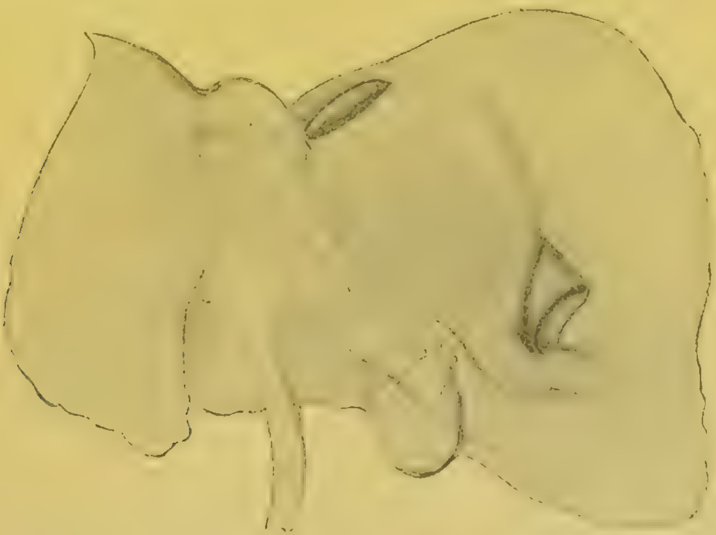


FIG. 27.—Cirrhotic liver with such enlargement of the Spigelian lobe as to form a complete tunnel for the inferior vena cava.

On section the liver is tough and, like a piece of conglomerate stone, is divided into areas of irregular size by grey, slightly gelatinous-looking, fibrous tissue. In extreme cirrhosis the interlobular tissue may have a red, somewhat spongy appearance and be rather depressed, as compared with the masses of liver tissue that are embedded in it and stand up on section on a higher level. This fibrous tissue is continuous with the depressed, more opaque areas in the capsule, and by its contraction has squeezed into prominence the more healthy parts of the liver, which thus form the nodules or hobnails.

This fibrosis spreads out from the medium-sized portal canals. The areas of liver substance thus enclosed vary in size, being usually from $\frac{1}{6}$ to $\frac{1}{4}$ inch in diameter, and contain six to ten lobules, each of which normally measures $\frac{1}{20}$ to $\frac{1}{16}$ inch in diameter. The liver substance is much paler than in health and has a yellowish-brown colour either from staining with bile or from fatty change. In the same liver the colour of different hobnails often varies, some being yellow, others brownish-red. This may be due to irregularity in the amount of fatty change.

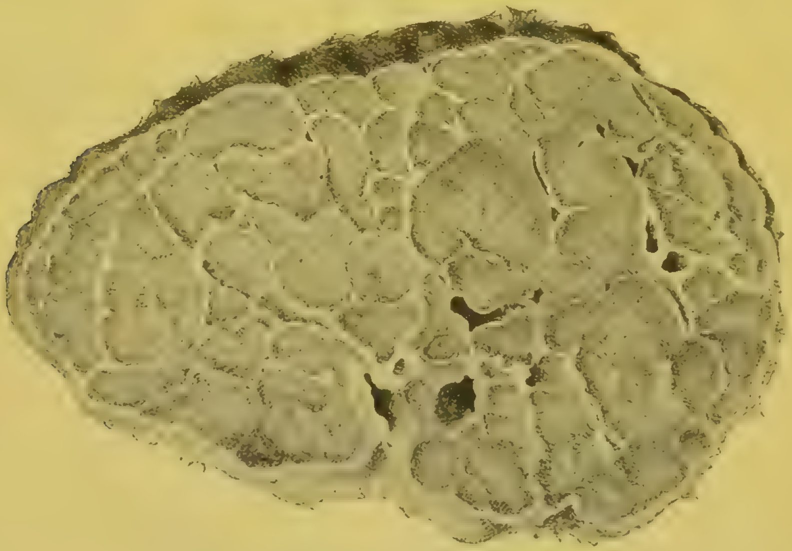


Fig. 28.—Section of multilobular cirrhotic liver shewing irregularity in the mesh of the fibrous trabeculae. (Drawn by Dr. E. A. Wilson.)

In rare instances haemorrhages are found in the interstitial tissue, in the hobnails, or in both. The liver cells may be compressed by the extravasated blood and sometimes shew necrosis. When the haemorrhage is widespread in the interlobular tissue and the hobnails have undergone fatty change, an appearance exactly like new-growth may be produced. The haemorrhages may possibly be ~~due to the~~ toxic ~~condition of the blood~~, but this is unlikely, since hepatic haemorrhages are rare in fatal cases of cirrhosis, in which death is usually due to toxaemia. In some instances it is due to fatty change in the cells in the hobnails removing the support of the contained vessels; Bonome[1] has suggested that endarteritis of the hepatic artery may produce necrosis of the liver cells and haemorrhages. As a coincidence I have seen numerous haemorrhages into the cirrhotic liver of a man who died from haematemesis. Occasion-

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[1] Bonome. *Arch. di biol.*, Firenze, 1899, liii, 319.

including ~~the~~ Kupffer's
Cells [Eppinger).

Eppinger, H. Wien. Klin. Wchnschr., 1922, 35 333 ally small thrombosed veins may be seen in the liver without any similar change in the portal vein or its larger branches. It has been suggested that thrombosis of the terminal branches of the portal vein may determine ascites, but this is seldom observed.

Histology.—In the early and more progressive stages there is small-celled infiltration in and around the portal spaces; these cells are due to hyperplasia of the existing connective tissues of Glisson's capsule and to some leucocytic invasion. In a well-marked case there is an irregular meshwork of connective tissue extending throughout the liver and dividing it up into variously sized islands of liver tissue. Inasmuch as several lobules are enclosed within the same fence of connective tissue, the term multilobular cirrhosis is applied. The number of lobules enclosed in these compartments varies; in some parts there are many, in other areas a single lobule or half a lobule is separated off from the rest.

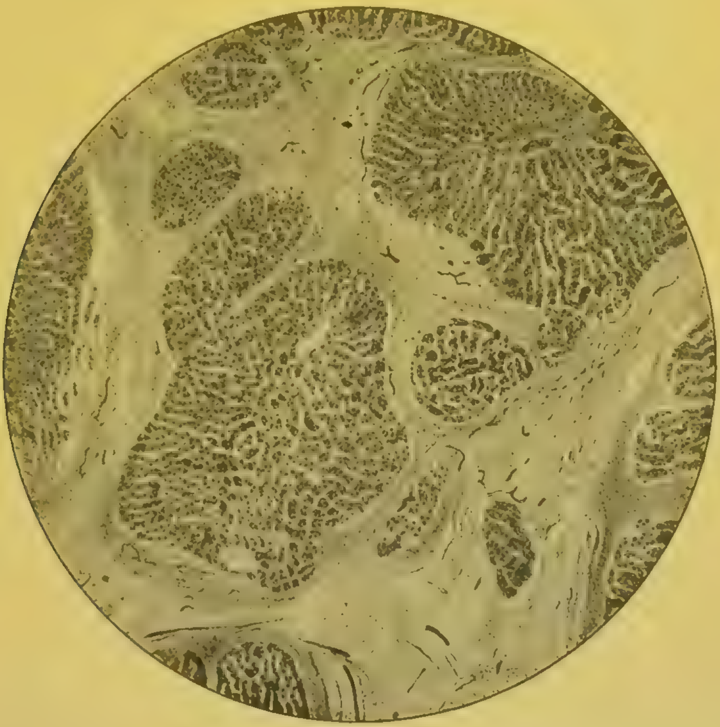

FIG. 29.—Multilobular cirrhosis. Well-formed fibrous tissue separating off masses of liver cells of various sizes. The normal arrangement of the liver cells is lost. In the fibrous tissue there are a few "newly-formed bile-ducts."

The French school considers that the fibrosis is not only portal, but also around the sublobular veins or bivenous (Sabourin[1]). It does not appear, at any rate to me, that there is fibrosis around the intralobular veins. At the margin of the lobules the fibrous tissue can be seen to surround bits of the lobule and thus to shave off groups of cells from the edge of the lobule. In some large cirrhotic livers, in which the meshwork is still multilobular as a whole, there are parts where it is more diffuse and approaches the unilobular type; this mixed cirrhosis is, from an anatomical point of view, a transitional stage to hypertrophic biliary cirrhosis; but it is common in ordinary portal cirrhosis when the disease is rapidly advancing.

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[1] Sabourin. *Rev. de méd.*, Paris, 1882, ii, 465.

When the morbid process is progressing rapidly the lobules are often actually invaded, so that a certain amount of intercellular fibrosis is superadded; Oertel[1] insists that the fibrosis is always intralobular as well as interlobular, and that what are described as lobules are not intact hepatic lobules. There are often areas of unilobular and intercellular cirrhosis in addition to the prevailing multilobular type. It is not justifiable or necessary, however, to construct a special clinical type of mixed cirrhosis to correspond to these microscopic appearances. The nature of the fibrous tissue varies according to its age, and whether the process is progressive or stationary. There is nearly always some well-formed fibrous tissue containing small round and spindle cells. In cases running a rapid course the amount of small round cells is increased, while in latent or stationary cases there may only be old fibrous tissue. The interstitial tissue contains numerous newly formed vessels derived from the hepatic artery, which are sometimes much dilated. The

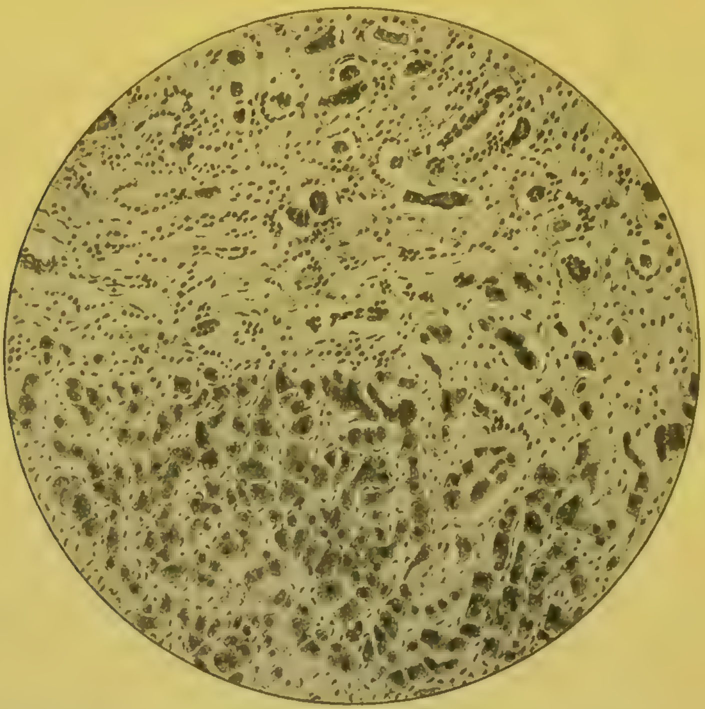

FIG. 30.—Multilobular cirrhosis. Shews invasion of a lobule by young connective tissue, so that there is local intercellular cirrhosis. Some of the liver cells thus isolated are hypertrophied and beginning to divide. The older connective tissue has some so-called new bile-ducts in its meshes. ×140.

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[1] Oertel. *Arch. Int. Med.*, Chicago, 1908, i, 385.

In some cases there is endophlebitis of the vessels running between the portal & the intralobular veins, this prevents compensatory hyperplasia of the liver cells & the liver is small (GÉRAUDEL).

GÉRAUDEL. *Arch. de méd. expér. et d'anat. path.*, Par., 1911, XXIII, 64

lymphatics undergo obliteration from endothelial proliferation, and to compensate for this fresh channels are formed (Oertel).

The increase in connective tissue, formerly assumed to consist solely of white fibrous tissue, has been shewn by modern histological methods to be partly due to an increase in the amount of elastic tissue.

Hohenemser[1] demonstrated elastic fibres in the connective tissue of portal cirrhosis; this has been confirmed by Melnikow-Raswedenkow,[2] Flexner,[3] and Fenzi.[4] The elastic tissue spreads out from the sheaths of the hepatic artery, portal vein, and bile-duct, and is also found in the capsule. More elastic tissue is found in portal than in biliary and mixed forms of cirrhosis. According to Carnot and Amet[5] there is considerable variation in the amount of elastic tissue present, but generally speaking there is more in old than in early cases of cirrhosis.

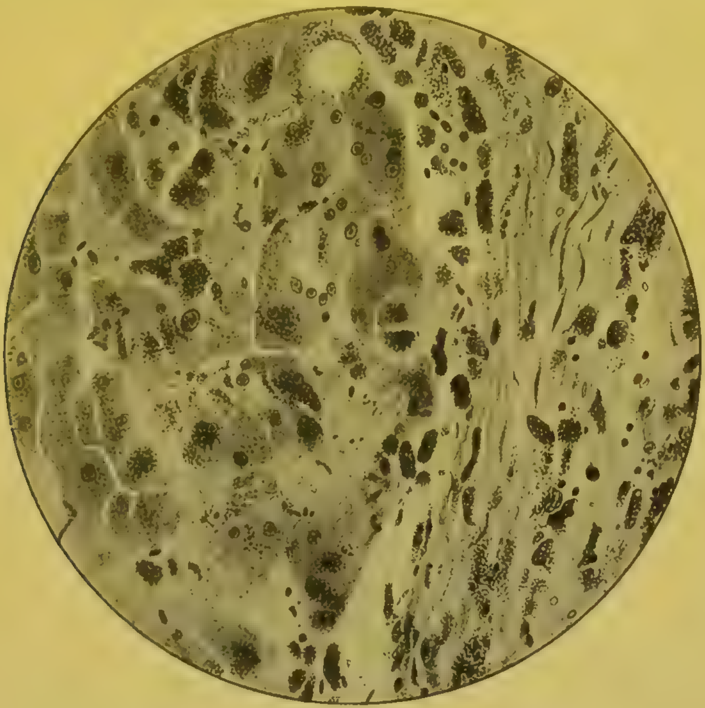

FIG. 31.—Cirrhosis in haemochromatosis. Section at margin of lobule. Shews haemosiderin as dark granules in the liver cells and also in the fibrous tissue surrounding the lobules. Prepared from Dr. Maude Abbott's case. (*Vide Trans. Path. Soc.*, Lond., 1900, li, 56.) ×220.

In pigmentary cirrhosis (*vide* p. 302), which may be due to more than one cause, the fibrous tissue may become occupied by opaque masses of pigment. In the cirrhosis of haemochromatosis the pigment is eventually liberated from the liver cells and passes into the connective tissues.

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[1] Hohenemser. *Virchows Arch.*, 1895, cxl, 192.
[2] Melnikow-Raswedenkow. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1899, xxvi, 526.
[3] Flexner. *Univ. Med. Mag.*, Phila., 1900–1901, xiii, 613.
[4] Fenzi. *Sperimentale*, 1904, lviii, 403.
[5] Carnot et Amet. *Arch. de méd. expér. et d'anat. path.*, Paris, 1906, xviii, 752.

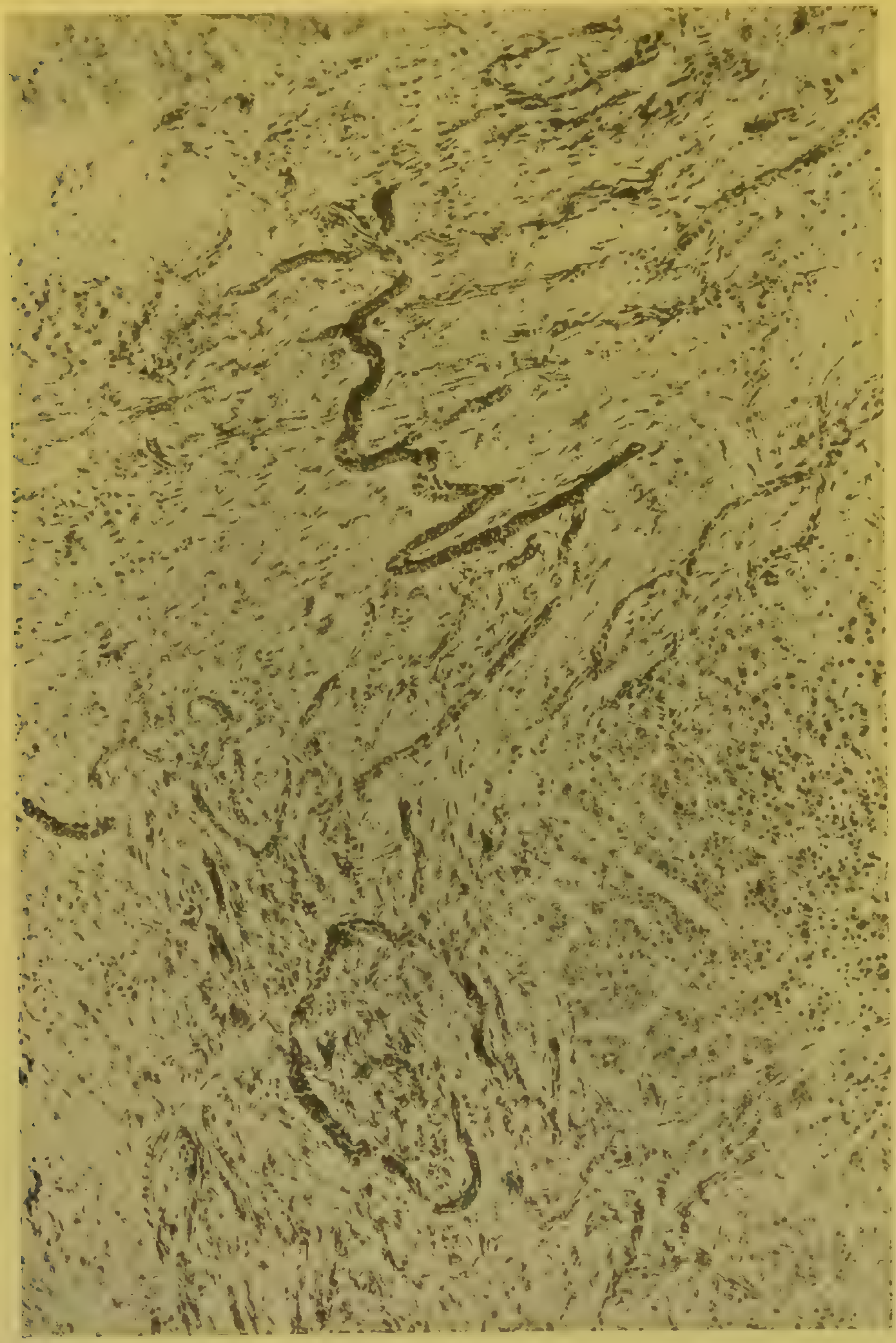

FIG. 32.—Photomicrograph shewing deeply stained pseudobile canaliculi branching and twisting in the fibrous tissue. The margin of a lobule is also shewn. From a case of multilobular cirrhosis. (Dr. H. Spitta.)

Pigmentation of the fibrous tissue in cirrhosis is also seen in malaria and in the extremely rare condition of cirrhosis anthracotica (*vide* p. 302).

Newly Formed Bile-ducts—Pseudobile Canaliculi—Bile-duct-like Structures.—In active cirrhosis the interstitial tissue shews as a prominent feature double columns of small cells which stain deeply and may shew mitotic figures. The cells are small and may be cubical, but are often elongated so as to lie in the long axis of the column; they are arranged, on much the same pattern as in a small bile-duct, around a potential lumen. Histologically they differ from normal interlobular bile-ducts in having either no elastic fibres, or a very imperfect development of them only (Flexner [1]). It is indeed open to doubt if similar structures containing a dilated lumen or masses of inspissated bile are exactly the same as these so-called new bile-ducts. The so-called new bile-ducts twist and branch in the interlobular connective tissue and form a rough network. The individual columns catch the eye as minute worm-like bodies under a low power. They often end in contact with liver cells, and it has therefore been thought that they (i) are derived from proliferation of liver cells, or (ii) that though derived from bile-ducts they form liver cells.

Special importance was formerly attached to the presence of these so-called new bile-ducts in hypertrophic biliary cirrhosis, but they may be just as prominent in common cirrhosis, and indeed are present in a number of conditions, such as local fibrosis around a hydatid cyst, gumma, tubercle, lymphadenoma, acute yellow atrophy, and other lesions which have in common destruction of the liver cells.

A considerable amount of discussion has arisen as to their nature. It has been thought that they are the normal bile-conducting channels running between the liver cells and the interlobular bile-ducts, which are left in a conspicuous position by the atrophy and recession of the liver cells; the brilliant staining of the cells has been explained by supposing that some proliferation of the cells normally lining the ducts in the interlobular tissues around the portal canals extends into the flat-walled ducts between the liver cells. Milne [2] argues that when these bile-duct structures are exposed in granulation tissue, their lining cells swell up in the same way that the lining cells of the air alveoli in chronic interstitial pneumonia do. In this way ducts lined with cubical epithelium are produced. Against the view that they are pre-existing bile channels is the fact that in senile or other forms of simple atrophy of the liver cells the appearance of so-called new bile-ducts is not seen. From their resemblance to small bile-ducts it has been widely thought that they are due to proliferation of pre-existing bile-ducts (Muir [3]). Another suggestion is that the liver cells become surrounded by the advancing fibrous tissue, which, so to speak, slices off columns of hepatic cells from the periphery of the lobules (*vide* Fig. 30). The liver cells then atrophy, become compressed, and are said to revert to the type of a tubular liver. This view as to the nature of the change is supported by some appearances, but is opposed to the facts that they stain deeply and are evidently proliferating actively. It has also been argued by Dreschfeld[1] and others that they are the result of active proliferation of the liver cells, and, as Hanot[2] believed, an attempt at compensatory hyperplasia. The healthy liver cells proliferate in order to compensate for the destruction of the hepatic tissues. Appearances strongly suggesting hyperplasia of the cells may be seen at the periphery of the lobules, and the columns of small cells may in fortunate sections be traced into continuity with a liver cell. Craven Moore[3] suggests that the bile-duct structures are derived from cells transitional between the liver cells and the bile-ducts. Since some doubt at any rate exists as to their being bile-ducts, it is more convenient to speak of them as the so-called new bile-ducts or pseudobile canaliculi.

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[1] Flexner. *Univ. Med. Mag.*, Phila., 1900, xiii, 617.
[2] Milne. *Journ. Path. and Bacteriol.*, Cambridge, 1909, xiii, 127.
[3] Muir. *Brit. Med. Journ.*, 1908, ii, 1169.

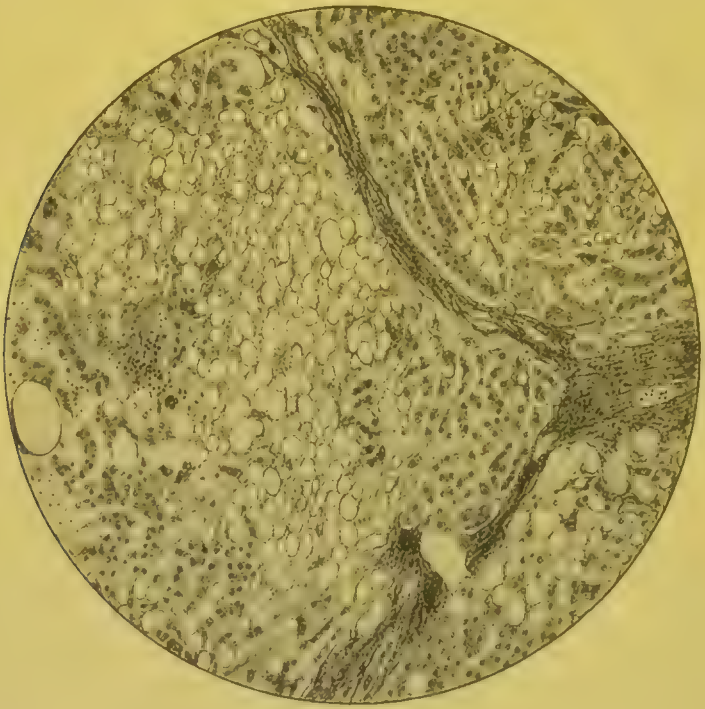

FIG. 33.—Slight multilobular cirrhosis with extensive fatty change in the liver cells. The vacuolation of the liver cells is irregular. Some groups of cells are unaffected, whilst others have hardly any protoplasm left. × 72.

The *liver cells* in the early stages shew degenerative changes which precede any interstitial reaction (Fiessinger[4]). There is a peculiar hyaline change which goes on to necrosis. These cells become surrounded by polymorphonuclear or endothelial leucocytes which absorb them (Mallory[5]). Active mitoses may be seen in the neighbouring cells,

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[1] Dreschfeld. *Journ. Anat. and Physiol.*, 1881, xv, 69.
[2] Hanot, V. *Gaz. des hôp.*, Paris, 1896, lxix, 897.
[3] Moore, F. C. *Brit. Med. Journ.*, 1908, ii, 1168.
[4] Fiessinger. *Arch. de méd. expér. et d'anat. path.*, Paris, 1908, xx, 313.
[5] Mallory. *Johns Hopkins Hosp. Bull.*, Balt., 1911, xxii, 73.

Mallory, Parker, and Nye found haemofuscin or haemosiderin in the hepatic cells in 7 per cent. of their cases of cirrhosis

, but varies greatly in different cases.

Mallory, Parker, and Nye. *Journ. med. Res.*, 1921, xlii, 461 a sign of regeneration on which Kretz[1] and MacCallum[2] lay stress. The normal trabecular arrangement in the lobule becomes disturbed and disappears. The intralobular vein may be absent or at one side of the enclosed mass of cells. In some cases, owing to backward venous pressure from cardiac failure, the capillaries between the columns of liver cells become dilated and separate single columns of cells from each other. In other cases the lymphatic capillaries are dilated and the lobule becomes oedematous. Fatty change is often present in a greater or lesser degree, and is the effect of the action of alcohol or some other toxic substance. It has been thought that fatty change is specially related to excess in beer and other sugar-containing beverages, but this assumption has not been proved (*vide* p. 195).

The degenerative changes are extremely prominent in cases which run a rapid or an acute course. The protoplasm may shew cloudy swelling and become granular, while the nucleus may stain badly or be obscured. The presence of granules of bile is seen in cases in which jaundice has existed, while in malaria, haemochromatosis, and in other examples of pigmented cirrhosis (*vide* p. 302) the liver cells are crowded with pigment and are markedly degenerated. The liver cells may shew siderosis quite apart from the existence of general haemochromatosis.

In 16 cases of cirrhosis Abbott[3] found pigment in the liver cells in 6 cases, and in 26 cases examined by Kretz[4] pigmentation existed in 14. ~~Since this pigment is probably due to the haemolytic action of intestinal bacteria, it is curious that it is not more constant in cirrhosis~~.

In the early stages of cirrhosis a majority of the liver cells, even those embedded in fibrous tissue, contain glycogen. This observation of Brault's[5] helps to explain the absence of glycosuria in cirrhosis. He suggests that in cirrhosis there may be excessive glycogen storage in the liver cells in order to compensate for the cell destruction.

Compensatory hyperplasia of the liver cells occurs in cirrhosis, The proliferation of the liver cells begins at the margin of the lobule, probably because the blood-supply is more copious there. The liver cells become larger, divide usually by direct nuclear division, but sometimes by karyokinesis, and by multiplying lead to deformity and to an increase in the size of the lobules and to the formation of actively growing masses of liver tissue. Turnbull and Worthington[6] describe two stages, (*a*) the regeneration-nodule, and (*b*) adenoma in which the cells are further removed from the normal hepatic cells. Many of the "hobnails" in a cirrhotic liver are of this nature. When the process is well marked, the condition is called nodular cirrhosis, or cirrhosis with adenoma. This process is important in compensating for the destruction of the liver cells and allowing the disease to become latent (Hanot and Gilbert[1]). As already pointed out, it has been thought that regeneration of liver tissue may be due to proliferation of the liver cells to form pseudobile-canaliculi (Dreschfeld, Hanot). Milne has recently denied that these "pseudobile canaliculi" are derived from hepatic cells or form them.

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[1] Kretz. *Wien. klin. Wchnschr.*, 1900, xiii, 271.

[2] MacCallum. *Journ. Amer. Med. Assoc.*, 1904, xliii, 649.

[3] Abbott, M. *Trans. Path. Soc.*, Lond., 1900, li, 79.

[4] Kretz, R. *Beitr. z. klin. Med. u. Chir.*, Wien, 1896, Heft 15. Centralbl. f. allg. Path. u. path.-Anat., 1897, viii, 620

[5] Brault. *Arch. de méd. expér. et d'anat. path.*, 1902, xiv, 453; *Bull. Soc. anat.*, Paris, 1901, lxxvi, 334.

[6] Turnbull and Worthington. *Arch. Path. Inst. London Hosp.*, 1908, ii, 63.

Gall-bladder and Bile-ducts.—The gall-bladder and larger bile-ducts do not shew any constant change to the naked eye. In some instances the walls of the gall-bladder are thickened from past or from chronic cholecystitis, and occasionally there is inflammation of the larger ducts. I have seen acute pneumococcic cholecystitis in latent cirrhosis (*vide* p. 613). In ascites the serous coat of the gall-bladder may be oedematous.

Gall-stones occur slightly more often in cirrhosis than in ordinary cases. Thus in ~~157~~ cases of cirrhosis examined after death at St. George's Hospital gall-stones were found in ~~23~~ or ~~14·6~~ per cent. In 209 male cases of cirrhosis gall-stones were found in 8 per cent, and in 41 females in 17 per cent (Klopstock[2]).

The small intrahepatic bile-ducts are usually healthy or shew little change. In some instances there is concomitant catarrhal cholangitis.

A young man, aged twenty-six years, with slight jaundice died from fatal haematemesis on Feb. 22, 1905. Eleven months previously he had had jaundice, from which he had completely recovered. The liver (72 oz.) was cirrhotic, the bile-ducts contained turbid milky bile, and microscopically (*vide* Fig. 34) there was pericholangitis and the formation of minute calculi in the intrahepatic bile-ducts. The spleen weighed 34 oz. The lymphatic glands in the portal fissure and around the pancreas were swollen, oedematous, and black from altered blood pigment.

In one case microscopic sections of a cirrhotic liver with some perihepatitis shewed cystic dilatation of the bile-ducts in a few of the portal spaces. It is indeed remarkable how extremely rare any dilatation of the small ducts is in cirrhosis, especially in view of the frequency of cysts in a granular kidney, which may be considered as homologous to hepatic cirrhosis (*vide* p. 444).

Portal Vein.—The intrahepatic branches of the portal vein are compressed, some are obliterated, and occasionally some are thrombosed. The trunk of the portal vein and its branches are dilated, and very occasionally varicosities are seen on its mesenteric radicles. The walls of the vein are thickened by periphlebitis and there is opacity of the intima from endophlebitis ; in extreme instances secondary calcification may occur in the intima. These changes are comparable to arterio-sclerosis and are connected with increased pressure in the portal vein, or possibly with the presence of poisons in the blood. Thrombosis of the trunk of the portal vein is probably more often associated with hepatic cirrhosis than with any other condition, but it is a rare complication ; it occurred in 6 out of 711 cases tabulated by Lissauer,[3] or 0·9 per cent,

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[1] Hanot et Gilbert. *Bull. Soc. méd. des hôp. de Paris*, 1890, 3. s., vii, 492.

[2] Klopstock. *Virchows Arch.*, 1907, clxxxvii, 111.

[3] Lissauer. *Ibid.*, 1908, cxcii, 278.

and in 10, or 3·3 per cent, of Langdon Brown's 334 cases of cirrhosis.[1]

Dilatation of the Communications between the Portal Vein and the General Systemic Veins.—This is important as providing a collateral circulation for venous blood which would otherwise have to force its way against the obstruction offered by the cirrhotic liver. This compensatory anastomosis is one of the factors which enable cases of multilobular cirrhosis to become latent or to appear cured. The normal communications between the portal vein radicles and the general systemic veins have been tabulated by Thomas[2] as (i) between the portal vein and the superior vena cava, (*a*) superficial, and (*b*) deep communications; and (ii) between the portal vein and the inferior vena cava, (*a*) superficial, and (*b*) deep. In both cases the deep communications are the more important. It is sufficient to consider them as (i) general and (ii) local.

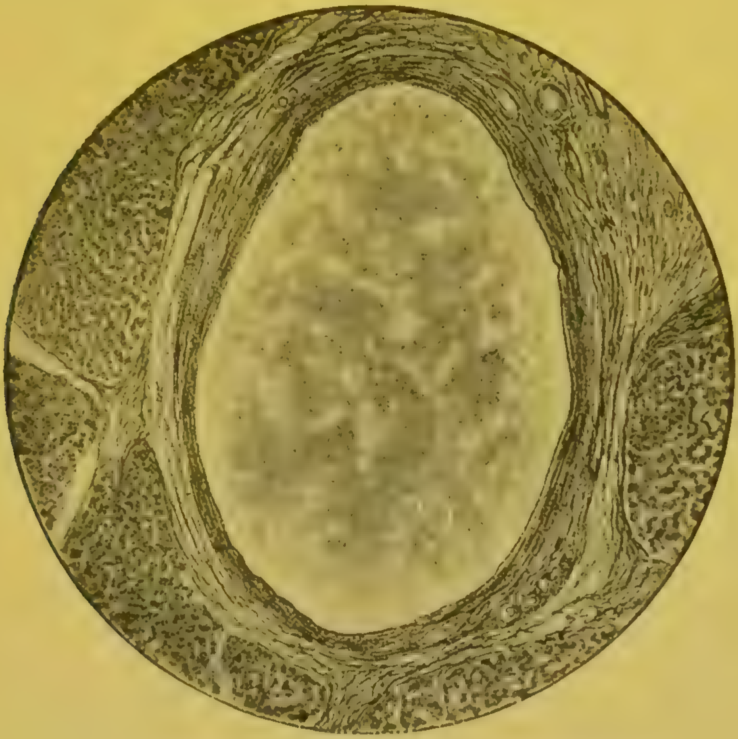

Fig. 34.—Dilatation of small bile-duct with pericholangitis in portal cirrhosis. × 24.

(i) *General Communications* between the retroperitoneal veins, opening into the lumbar and azygos veins, and the veins of the peritoneum and of the intestines occur especially where, as in the case of the duodenum, pancreas, ascending and descending colon, areas drained by the portal vein are bound down to the abdominal parietes. The veins in the capsule and perirenal fat of the left kidney anastomose to some extent with the veins of the descending colon (Tuffier and Lejars [1]), and venous trunks may put the renal vein itself into communication with the colica sinistra vein. The veins of the descending colon may communicate with the spermatic plexus, and this has been thought to favour the development of varicocele on the left side (Bennett [2]). Compensatory dilatation of this anastomosis has been recorded, especially in Livierato's [3] case, in which the left spermatic vein was 30 inches long, tortuous, and greatly dilated; but in general it is of little or no importance. It has not appeared to me that varicocele is specially common or marked in cirrhosis.

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[1] Brown, Langdon. *St. Barth. Hosp. Rep.*, 1901, xxxvii, 62.

[2] Thomas. "Beiträge zur Differentialdiagnose zwischen Verschluss der Pfortader und der unteren Hohlader." *Bibliotheca Medica*, 1895.

A communicating branch between the splenic and the left renal veins has been seen, and in Jacobson's [4] case of compensated hepatic cirrhosis, in which death occurred from uraemia due to granular kidneys, a vein as large as the thumb ran from the left renal vein into the trunk of the portal vein. Virchow [5] observed a somewhat similar anastomosis between the splenic and the azygos veins.

In some cases of cirrhosis the greater part of the parietal peritoneum, more especially on the posterior wall and upper part of the abdomen, is markedly injected and in some areas resembles a "claret stain" on the skin. The significance of this, and also the fact that it is not due to a terminal peritonitis, are shewn by the absence of this injection from the peritoneum covering the free coils of the small intestine. This subperitoneal plexus of anastomoses between the branches of the portal vein and the inferior vena cava was specially described by Retzius,[6] after whom it is sometimes called. Peritoneal adhesions around the liver, stomach, omentum, or spleen may become markedly vascular and thereby assist the collateral circulation.

This hint on the part of nature has been taken in a surgical sense in the operation (Talma-Morison) for the relief of ascites by the production of artificial peritoneal adhesions (*vide* p. 260).

(ii) *Local.*—(*a*) Around or in connexion with the liver: An internal porto-caval anastomosis develops in the lobules of the liver between the branches of the portal vein and the intralobular vein. Communications may develop between the veins in the substance and capsule of the liver and the phrenic and intercostal veins, where the liver and diaphragm are uncovered by peritoneum, *i.e.* between the layers of the coronary ligament and its lateral fringes, the lateral ligaments.

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[1] Tuffier et Lejars. *Arch. de physiol.*, Paris, 1891, xxiii, 41.
[2] Bennett, W. H. *On Varicocele*, p. 43, 1891.
[3] Livierato. *Riforma med.*, Napoli, 1911, xxvii, 505.
[4] Jacobson, G. *Arch. gén. de méd.*, 1893, i, 353.
[5] Virchow. Quoted in Frerichs' *Diseases of the Liver*, vol. ii, p. 41. Transl. New Syd. Soc., 1861.
[6] Retzius. *Ztschr. f. Physiol.*, 1833, v, 105.

Hanganutz has collected 6 cases
of persistence, total or partial
of the umbilical vein in what
he terms Cruveilhier-Baumgarten's
cirrhosis

HANGANUTZ, M. Presse méd., Paris, 1922, XXX, 732.

In a man aged fifty-eight years, who died under the care of Dr. C. Ogle in St. George's Hospital, the left internal mammary vein shewed a local varicose dilatation behind the sternum, where during life a loud murmur had been heard, and then turned inwards into the falciform ligament and ran into the left lobe of the liver and communicated with the left branch of the portal vein. The liver was small and cirrhotic (28 oz.).

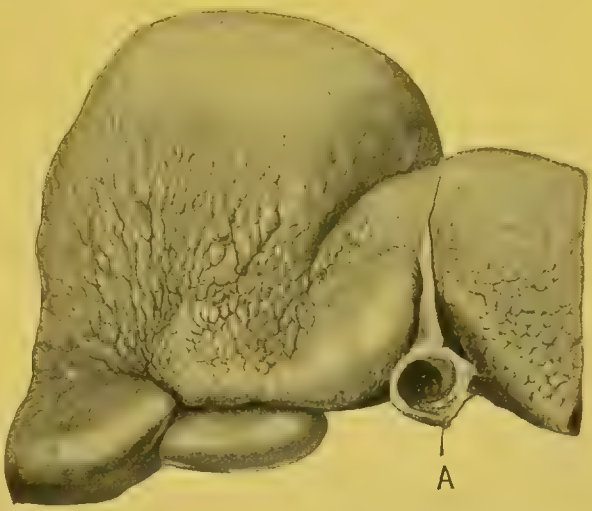

FIG. 35.—A cirrhotic liver with a large parumbilical vein (A) in the falciform ligament. (St. George's Hosp. Museum, Series ix, 174M.)

The falciform ligament of the liver may contain a large vein which runs to the umbilicus and may communicate there with the veins of the abdominal wall, and so establish a communication between the portal vein in the transverse fissure of the liver and the deep epigastric and external iliac veins. This vein is comparable with the epigastric vein of cold-blooded air-breathing vertebrates, such as the frog. This vessel may be a greatly dilated parumbilical vein, which, according to Luschka, normally puts the portal and epigastric veins into communication, and runs alongside of the obliterated umbilical vein. It was originally stated by Rokitansky and Bamberger that this vein was the umbilical vein, which had become pervious again and, so to speak, had reverted to its fetal function of carrying blood, with the difference that the blood runs away from, instead of to, the liver. Sappey[1] insisted that the large vein found in the falciform ligament in some cases of cirrhosis was always independent of the umbilical vein; but this is not in accord with cases quoted by Champneys.[2] This vein may be extremely large; Wilks[3] mentions a case in which it was as big as the portal vein, and in Sappey's case and the one figured here it admitted the little finger. This big vein is probably not so infrequent as would appear from the recorded cases, for the vein collapses after death and is not seen unless the falciform ligament is specially examined. I have seen it full of ~~carcinoma~~ growth in a case of carcinoma with cirrhosis.

As a result of the anastomosis thus opened up the veins of the abdominal wall, especially around the umbilicus, may become dilated and prominent. When marked, this condition is spoken of as a "caput

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[1] Sappey. *Bull. Acad. de méd.*, 1859, xxiv, 270.
[2] Champneys. *Journ. Anat. and Physiol.*, 1872, vi, 417.
[3] Wilks. *Pathological Anatomy*, p. 467, 1889.

Medusae," but is hardly ever so well developed as the caput Medusae produced by mechanical obstruction of the inferior vena cava. There is a difference in the situation of these two collateral venous circulations in the abdominal wall; that due to portal obstruction is most marked around the umbilicus, while that seen in the obstruction of the inferior vena cava runs from the middle of the groins to the costal arches and avoids the umbilicus (*vide* p. 249).

It may be pointed out that in healthy subjects the round ligament at its junction with the right branch of the portal vein is pervious for a distance of ½ to 1½ inches and then becomes occluded. This channel is no doubt utilised in the formation of a collateral and compensatory circulation.

In very rare instances the ductus venosus, which in the fetus runs directly from the umbilical vein into the inferior vena cava, is persistent, and thus provides a direct communication between the portal vein and the inferior vena cava.

In an interesting case of hepatic cirrhosis with obstruction of the superior vena cava described by Duckworth and Garrod[1] there were greatly dilated veins under the skin of the abdomen, the blood running into them in both directions, but it is noteworthy that there was an absence of these veins around the umbilicus.

In ordinary cirrhosis extensive ascites may so compress the inferior vena cava as to lead to the development of a caput Medusae comparable to that seen when there is obstruction to the blood flow through the inferior vena cava. An extremely rare complication of cirrhosis is thrombosis of the inferior vena cava, which would produce the same conditions.

Pheasants[2] collected 8 cases. Councilman[3] met with a terminal streptococcic infection in cirrhosis in which the retroperitoneal glands and tissues were suppurating and had thus caused thrombosis of the inferior vena cava.

Occasionally there may be a kind of belt of dilated cutaneous venules over the line of attachment of the diaphragm. This, however, is not pathognomonic of portal obstruction, as has sometimes been thought; for it occurs in emphysema, bronchitis, and sometimes in elderly men without at any rate any manifest morbid lesion (*vide* p. 229).

(*b*) At the terminations of the intra-abdominal parts of the alimentary canal: The veins of the oesophagus, which open into the azygos veins, communicate at the cardiac orifice with the gastric veins and so with the portal vein. This communication may become so developed as to produce varicosities at the lower end of the oesophagus, or "oesophageal piles," and from ulceration or rupture may be the source of severe haemorrhage. Though usually developed to a certain degree, it is only occasionally that there are large varicosities. This may be explained on

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[1] Duckworth and Garrod. *St. Barth. Hosp. Rep.*, 1896, xxxii, 71.

[2] Pheasants. *Bull. Johns Hopkins Hosp.*, 1909, xx, 292.

[3] Councilman, W. T. *Trans. Assoc. Am. Phys.*, 1896, xi, 213.

the ground that there are valves guarding the entrance of the gastric vein (Macalister [1]), which would tend to prevent reflux of blood from the portal into the gastric veins.

It should be noted that oesophageal varix may occur without cirrhosis of the liver; cases of fatal haemorrhage have occurred from this cause in a child and in a boy of seventeen.[2]

The coronary vein of the stomach communicates with the phrenic vein, and it would be natural to anticipate that the branches of the gastric veins would be often dilated. This is very seldom observed, though it is possible that it is overlooked. The walls of the vein may undergo chronic inflammation, and from fibrotic atrophy shew local dilatations. These become adherent to the mucosa, and the raised area may, as the result of trauma, toxic irritation, or infection, become ulcerated. The resulting haemorrhage may, as in two cases of cirrhosis recorded by Letulle,[3] be fatal.

Varicosity of the gastric veins and fatal haematemesis may occur in the absence of any hepatic disease (Barr,[4] Lancaster [5]).

Dilatation of the anastomosis between the superior haemorrhoidal vein opening into the inferior mesenteric, on the one hand, and the middle and inferior haemorrhoidal, which pour their blood into the internal iliac vein, on the other hand, may also occur. It would if excessively developed lead to piles, and it has been assumed that piles are common in cirrhosis. This, however, is not the case, and constipation is a far more frequent and important factor than cirrhosis in the production of piles.

It is possible that the compensatory effects of these anastomotic channels have been somewhat overestimated. Thus the dilated venous channels may be absent in latent cirrhosis fatal from other causes, and may be well developed in cases dying with ascites. In the latter instance the compensatory process, though considerable, cannot be regarded as having been successful.

In an examination of 78 autopsies of cases with cirrhosis, Sears and Lord [6] found this dilated anastomosis in 29 cases, being most often present when the cirrhotic process was most advanced. But in 22 out of the 29 cases there was ascites.

Bad Effects and Results which may depend on Excessive Development of this Venous Anastomosis.—The development of a free communication between the portal and general systemic veins, while it relieves portal engorgement, may lead to bad effects. In the first place, if the communications are extensive, it necessarily follows that a considerable quantity of blood carrying the products of digestion, instead of going to the liver, will pass directly into the general circulation and set up toxaemia. If the anastomosis is very free, the condition would resemble that in Eck's fistula, or the experimental communication between the inferior vena cava and the portal vein, in which all the portal blood passes into the inferior vena cava. A most elaborate and interesting series of experiments on these lines was carried out by Hahn, Massen, Nencki, and Pawlow.[1] In animals thus treated, effects equivalent to those of hepatic insufficiency naturally resulted, and when a protein diet was adopted, symptoms comparable to those of uraemia developed, which were regarded as due to the presence of carbamic acid in the blood. In man excessive development of the collateral circulation may lead to a toxaemic condition verging on uraemia. This has been insisted on by Stockton,[2] and it is interesting to note that one of Morison's[3] patients, after the operation for the surgical relief of ascites, was alternately excited and depressed for three weeks after the operation. Possibly a very free collateral anastomosis between the portal and general venous systems, while relieving portal engorgement, may lead to arteriosclerosis and granular kidneys. In cases in which the two conditions of renal and hepatic fibrosis are combined the patients usually die from renal disease with latency of the hepatic cirrhosis. This was the termination of Jacobson's[4] remarkable case, in which a vein as large as a thumb ran between the portal vein and the left renal vein.

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[1] Macalister's *Anatomy*, p. 431, 1889.
[2] Graham. *Trans. Assoc. Am. Phys.*, 1896, xi, 215.
[3] Letulle. *Presse méd.*, Paris, 1898, ii, 313.
[4] Barr. *Lancet*, 1889, ii, 1226.
[5] Lancaster. *Trans. Clin. Soc.*, Lond., 1897, xxx, 32.
[6] Sears and Lord. *Boston Med. and Surg. Journ.*, 1902, cxlvii, 285.

In the second place, haemorrhage may occur from rupture of the dilated venous anastomoses. This generally occurs into the gastro-intestinal tract, but in very rare instances, of which examples will be given, haemorrhage may take place into the abdominal wall or into the peritoneal cavity. Haematemesis not very infrequently results from rupture of an ulcerated varicose vein in the oesophagus, while slight bleeding from piles may go on for a considerable time.

The occurrence of a haematoma in the anterior abdominal wall close to the round ligament, though rare, is another illustration of a pathological event in the course of this compensatory mechanism, and is analogous to haematemesis from dilated oesophageal veins.

A case is recorded by Lefas[5] of a haematoma the size of a tangerine orange to the left of the middle line in a man, aged fifty-five, with cirrhosis. There is an interesting specimen (380·8) in the Museum of the College of Medicine, Newcastle-upon-Tyne, shewing a large haematoma in the abdominal wall close to the round ligament, which contains a number of dilated veins. The liver

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[1] *Arch. f. exper. Path. u. Pharmak.*, 1893, xxxii, 161.

[2] Stockton. *Journ. Am. Med. Assoc.*, 1889, xiii, 267; 1901, xxxvii, 817.

[3] Morison. *Lancet*, 1899, i, 1426.

[4] Jacobson, G. *Arch. gén. de méd.*, 1893, i, 353.

[5] Lefas. *Bull. Soc. anat.*, Paris, 1902, lxxvii, 586.

shewed ordinary portal cirrhosis. Thomas[1] records a similar haematoma in the abdominal wall in the middle line, but below the umbilicus.

In extremely rare instances trauma may lead to rupture of dilated veins in peritoneal adhesions and so to extensive haemorrhage into the peritoneum.

A man, aged forty-five, received a blow on the abdomen, and died in St. George's Hospital after being in a condition of collapse for eleven hours. The necropsy shewed cirrhosis of the liver (78 ounces) with marked engorgement of the veins in the substance of the liver, so that the organ had a naevoid appearance. The abdominal cavity was full of blood. The spleen was enlarged, and had haemorrhage into its substance, but there was no rupture of its capsule. There was laceration of some vascular adhesions, from which the blood probably came. Microscopically the liver shewed latent multilobular cirrhosis of old date with hardly any recent proliferation of the connective tissues.

From the anastomoses between the veins of the capsule of the left kidney and the branches of the portal vein in the mesocolon congestion of the vessels of the left kidney has been described (Gilbert and Villaret[2]) and may thus account for haematuria.

Hepatic Artery The ~~hepatic artery~~ is usually considerably enlarged, as it is thought to supply the added fibrous tissue with blood. Chronic endarteritis is not a special feature of ordinary cirrhosis, but it is seen in the cirrhosis of ~~due to~~ haemochromatosis and sometimes in cirrhosis in syphilitic patients and in parasyphilitic cirrhosis.

The *portal lymphatic glands* are not enlarged in uncomplicated cases. I have seen them swollen, oedematous, and black from blood pigment in fatal haematemesis (*vide* p. 208), and on several occasions pigmented by carbon.

The Spleen is enlarged in portal cirrhosis, though not so markedly as in hypertrophic biliary cirrhosis. According to Klopstock[3] it is enlarged in 80 per cent of the cases.

In Kelynack's series of 84 cases of cirrhosis the average weight of the spleen was 12·9 ounces. In 147 cases of cirrhosis examined at St. George's Hospital the average weight of the spleen was 10 ounces. So that if the weight of the normal spleen be taken at 7 ounces, there is seen to be a very definite increase in cirrhosis. The weight of the spleen varied between the two extremes of 2 ounces, in a case of latent cirrhosis, and 48 ounces, in a man who survived thirty months after the operation for producing artificial peritoneal adhesions had been performed. This weight is very exceptional in portal cirrhosis, though not in hypertrophic biliary cirrhosis.

In cases in which there has been fatal and very profuse haematemesis or very rapid ascites, the spleen may be small.

In latent cirrhosis the enlargement and increase in the weight of the spleen are much less than in cases in which death has been directly due to cirrhosis.

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[1] Thomas, Pierre. *Lyon méd.*, 1901, xcvii, 289.

[2] Gilbert et Villaret. *Arch. gén. de méd.*, 1906, xviii, 422.

[3] Klopstock. *Virchows Arch.*, 1907, clxxxvii, 111.

In 74 cases in which cirrhosis, though present, was not the cause of death, the average weight of the spleen was 9 ounces; while in 73 cases in which death was referred to the effects of cirrhosis, the average weight of the spleen was 11·5 ounces.

The fact that the spleen is bigger and heavier in cases dying from the effects of cirrhosis than in latent cirrhosis may be explained as depending on two factors which come into play in progressive cirrhosis—viz. (1) general toxaemia, and (2) increased pressure in the portal vein—but are either absent or largely obviated in latent compensated cirrhosis.

In very exceptional cases, of which I have seen two, the spleen is greatly enlarged and the liver shews slight and very old fibrosis. One of these cases was published by R. S. Trevor,[1] the other was a case of chronic splenic anaemia. This combination is not Banti's disease, in which cirrhosis supervenes as a terminal phenomenon of chronic splenic anaemia. It appears indeed to be the reverse of Banti's disease, and to be a continuation and exaggeration of morbid changes in the spleen in latent or practically cured cirrhosis.

Morbid Appearances of the Spleen.—The capsule of the spleen is often thickened and shews perisplenitis. This may be local, the thickened area being a lamellar fibroma, a condition frequently seen in splenic enlargement from any cause, or it may be general, and then usually forms part of a general chronic peritonitis.

Perisplenitis was present in 43, or 33 per cent, of 131 cases of cirrhosis analysed by Yeld;[2] and in 23, or 29·5 per cent, of the 78 cases recorded by Sears and Lord.[3]

The spleen is frequently adherent to the peritoneum covering the diaphragm, abdominal wall, and stomach. When the spleen and liver are both considerably enlarged, the left lobe of the liver may overlap the spleen. Though this is more likely to occur in hypertrophic biliary cirrhosis, it may occasionally be seen in multilobular cirrhosis.

On section the organ is sometimes firmer than natural and fibrosed; in other cases, probably as the result of terminal infections, it is soft or even diffluent. Occasionally areas of extravasation of blood are seen.

In a case of cirrhosis which survived the Talma-Morison operation for two and one-half years, the spleen was greatly enlarged, weighing 48 ounces, and contained numerous gritty spots. Microscopically these areas shewed fibrosis surrounding calcareous granules and particles which turned blue on being treated with ferrocyanide of potassium and hydrochloric acid (*vide* Fig. 36). There was also considerable hyperplasia of the endothelial cells lining the sinuses.

Microscopic Appearances.—There is dilatation of the blood-vessels generally, which spreads to the capillaries in the Malpighian bodies and

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[1] Trevor, R. S. *Trans. Path. Soc.*, 1903, liv, 302.

[2] Yeld, R. A. *St. Barth. Hosp. Rep.*, 1898, xxxiv, 215.

[3] Sears and Lord. *Boston Med. and Surg. Journ.*, 1902, cxlvii, 285.

and peri-arterial
fibrosis. | ^

is followed by secondary atrophy of the lymphoid tissue in the Malpighian bodies. Some difference of opinion exists as to proliferation of the splenic pulp. Oestreich[1] described it in early cases, but neither Sieveking[2] nor Azzurrini[3] confirmed this. From examination of 38 cases Christian[4] found increase of connective tissue due to proliferation of the reticular tissue of the pulp, but little or no increase in the white fibrous and elastic tissues. Fibrosis of the spleen due to increase in the trabeculae occurs in cases of some standing.

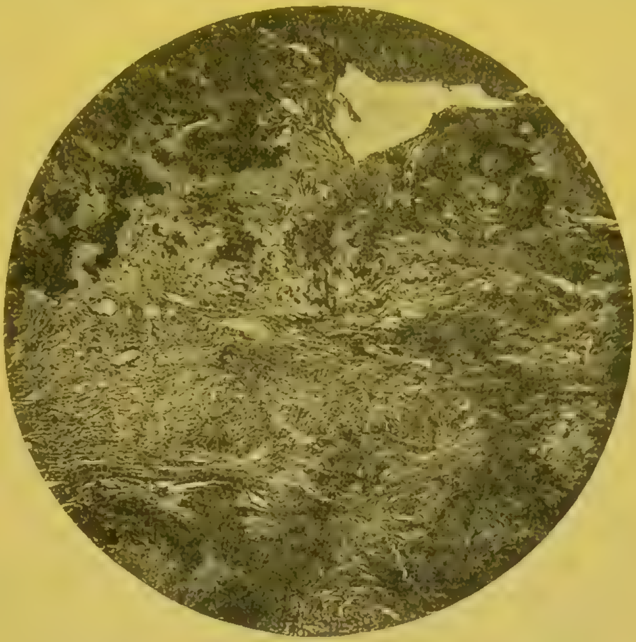

FIG. 36.—Photomicrograph shewing fibrous area in the spleen. Around it are dark masses which stained blue with ferrocyanide of potassium and hydrochloric acid. (By Dr. S. G. Penny.)

The Cause of Splenic Enlargement in Hepatic Cirrhosis.—There are two views as to the causation of the splenic enlargement.

(I) The mechanical view: that it is due to backward pressure and chronic venous engorgement of the organ brought about by the portal obstruction existing in the liver. In favour of this is the fact that the enlargement may diminish after profuse gastro-intestinal haemorrhage or diarrhoea. The manifest objection to this view is that in the chronic venous engorgement of heart disease the spleen, though firmer than natural, is not increased in weight. This is well brought out in a comparison drawn up by Kelynack[5] between the weights of the spleen (i) in 84 cases of cirrhosis and (ii) in 56 cases of nutmeg liver, in cases of heart disease uncomplicated by any febrile or other condition likely to affect the size of the organ.

	AVERAGE WEIGHT.		
	Males.	Females.	Both Sexes.
"Hepatic" spleen	14·25	11·62	12·93
"Cardiac" spleen	7·32	7·32	7·32

Some explanation of this difference might be found in the fact that the liver would act as a kind of buffer when the backward pressure is due to heart or lung disease, and so protect the spleen from excessive venous engorgement, whereas in cirrhosis the spleen is nearer to the venous obstruction. In support of this argument of Foxwell's[1] it may be added that in thrombosis of the splenic vein the spleen is very markedly enlarged. But, on the other hand, the splenic enlargement is much more marked and may precede any manifest change in the liver in hypertrophic biliary cirrhosis,[2] in which the obstruction to the passage of blood through the liver is much less than in portal cirrhosis. It appears, therefore, that backward pressure does not, at any rate completely, explain the splenic enlargement.

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[1] Oestreich. *Virchows Arch.*, 1895, cxlii, 285.
[2] Sieveking. *Centralbl. f. allg. Path. u. path. Anat.*, Jena, 1894, v, 1017.
[3] Azzurrini. *Arch. di biol.*, Firenze, 1902, lvi, 597.
[4] Christian. *Journ. Am. Med. Assoc.*, 1905, xlv, 1615.
[5] Kelynack. *Birmingham Med. Rev.*, 1897, xli, 86.

(II) The toxaemic view: that the spleen is enlarged as a result of poisons circulating in the blood and reaching it through the splenic artery, and that there is an active inflammatory swelling of the spleen like that seen in bacterial and some toxic conditions. In favour of this view is the absence of splenic enlargement in latent cirrhosis, in which the disease is arrested or compensated for. The toxaemia responsible for the enlargement might be either the same as that producing hepatic cirrhosis or it might be secondary to hepatic insufficiency, the cirrhotic liver allowing poisons, which should normally be arrested by it, to pass into the general circulation. This question is a difficult one. The spleen and liver react in different degrees to haemic intoxication, the spleen usually to a greater extent. Probably the spleen may suffer to some extent at the same time as the liver in early cirrhosis, if the poison, reaching the liver, as it generally does, from the portal system escapes into the general circulation. But when the liver becomes unable to stop poisons and general toxaemia supervenes, the conditions favourable for splenic enlargement are greatly increased.

Histologically Oestreich[3] found proliferation of the splenic pulp and concluded that it was due to toxins reaching the organ by the splenic artery.

To sum up: Splenic enlargement in cirrhosis probably depends primarily on poisons setting up hyperplasia and inflammatory softening of the organ so that it is readily overdistended with blood. Though chronic venous engorgement alone is not enough to cause enlargement of the spleen, it favours the stagnation of poisons and irritating bodies, and, further, it supplies blood to distend the softened splenic blood sinuses. Hence it may be concluded that the enlargement of the spleen is primarily due to toxic action and is aided by increased venous pressure in the portal system.

Relation between the Size of the Liver and that of the Spleen.—It might be surmised that in portal cirrhosis there would be a general relation between the size of the liver and that of the spleen. Examination of cases and statistics shews that this is a complicated question. Thus, in some cases, especially those running a rapid course and those

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[1] Foxwell, A. *The Enlarged Cirrhotic Liver*, p. 6, 1896.

[2] Compare F. P. Weber. *Edin. Med. Journ.*, 1897, N.S. ii, 579.

[3] Oestreich. *Virchows Arch.*, 1895, cxlii, 285.

presenting a mixture of portal and biliary cirrhosis, the liver and spleen are both enlarged. On the other hand, in latent or compensated cirrhosis the liver is considerably increased in size, being bigger than in cases fatal from the effects of cirrhosis, whilst the spleen is either of normal size or but slightly heavier than normal. Again, in cases fatal from cirrhosis the liver is sometimes smaller than natural, while the spleen is almost constantly larger and heavier than normal.

In order to see if any relation exists between the weight of the liver and of the spleen in cirrhosis the following percentages were extracted from a number of cases examined at St. George's Hospital.

In 43 male cases in which the liver weighed over 60 oz. (averaging 79·6 oz.), the spleen averaged 11 oz.; while in 7 males in which the liver weighed under 50 oz. (averaging 39·4 oz.), the spleen weighed 9·4 oz. In 11 female cases in which the liver weighed over 55 oz. (averaging 73·5 oz.), the spleen weighed 7·6 oz.; while in 9 cases in which the liver weighed less than 45 oz. (averaging 35·4 oz.), the spleen weighed as much as 9·8 oz. Therefore, in male cases of cirrhosis, whether fatal from the disease or from other causes, large livers were associated with a more considerable enlargement of the spleen than is seen when the livers are small. But in a corresponding collection of female cases the spleen was heavier in the series of small livers than in the large liver series.

A consideration of the whole subject shews that the weight of the spleen has no constant relation to that of the liver, and that it depends on other factors, which may or may not be associated with increased weight and size of the liver.

Morbid Appearances in other Viscera.—Some writers regard hepatic cirrhosis as a disease which is not strictly limited to the liver, but as part of a general change, and in this respect comparable to the condition of red granular kidneys which is a local manifestation of a general vascular change—arteriosclerosis. Portal cirrhosis might in the same way be considered to be part of a general change in the alimentary system, in which the spleen, the intestines, and the pancreas share. Klippel and Lefas[1] have pointed out that in some instances of cirrhosis the changes may be more advanced in the pancreas than in the liver. This relation of cirrhosis to a general cause is also seen in haemochromatosis, in which, as a secondary result, cirrhosis of the liver and pancreatic fibrosis may occur (*vide* p. 303).

The Oesophagus.—There may be chronic oesophagitis with general thickening of the mucous membrane, which in places shews localised elevations due to hyperplasia of the epithelium—small corns. The longitudinal veins running in the submucosa of the oesophagus in its lower 3 or 4 inches are often dilated and varicose (*vide* p. 212). Inflammatory changes in the varicose veins lead to adhesions between the veins and the mucous membrane, and elevations on the surface of the oesophagus thus result. Ulceration may then occur and give rise to very profuse haemorrhage.

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[1] Klippel et Lefas. *Rev. de méd.*, Paris, 1903, xxiii, 23.

Stomach.—Portal obstruction induces chronic venous engorgement of the stomach which nearly always shews chronic gastritis, mammillation of the mucous membrane and pigmentation, especially near the pylorus. There is occasionally dilatation of the gastric veins close to the cardiac orifice, but a varicose condition of the veins of the stomach is very rare. Cases of haematemesis depending on ruptured varicose gastric veins have been described (*vide* p. 271). Acute gastritis sometimes supervenes on the chronic gastritis, and small multiple ulcers which are quite superficial may then occur. A single chronic gastric ulcer is, however, rare in cirrhosis of the liver.

The Intestines. — There may be signs of chronic enteritis, such as thickening of the mucous membrane with tenacious mucus on its surface. Mere engorgement is not of very much importance after death, inasmuch as it may be largely due to stagnation of venous blood, from a dependent position of the intestinal coils. The mucous membrane may be pigmented from chronic catarrhal enteritis and venous engorgement. This is often more marked in the duodenum and the upper part of the jejunum than elsewhere. Occasionally the mucous membrane is oedematous. The subperitoneal tissues of the intestine may also be oedematous, either with or without oedema of the mucous membrane. Dilated venules and submucous haemorrhages are sometimes seen.

The small and large intestines become diminished in length in severe cases of portal cirrhosis; instead of 25 feet, the small intestine may only measure 15 feet, while the colon may be shortened in a corresponding or even in a greater degree. This fact, originally observed by Bright, has been explained as due to chronic enteritis, to concomitant chronic peritonitis, or to chronic inflammation of the radicles of the portal vein. It is important in that it diminishes the area of the mucous membrane from which absorption can take place. In rare instances the colon may be oedematous.

A little girl aged ten years was admitted to St. George's Hospital with a temperature of 104°, diarrhoea, and a swollen abdomen; the next day the abdomen was more distended and it was thought that perforation of a typhoid ulcer might have occurred. Laparotomy revealed a clear ascites and a cirrhotic liver. The patient died the next day. At the necropsy the liver (12 oz.) shewed extreme portal cirrhosis; there was no portal thrombosis. The mucous membrane of the colon was enormously swollen from oedema, and shewed a little follicular ulceration. There was no haemorrhage into the mucosa. There was no tubercle. An acute infection probably accounted for the high temperature and the oedema of the colon (*vide* also p. 180).

The rectum occasionally shews dilated veins, but~~, contrary to what might be expected,~~ piles are rare (*vide* p. 213). Occasionally there is intense congestion of the rectum with small ulcers which may give rise to considerable haemorrhage.

Changes in the *pancreas* are extremely frequent.

, which is of the adult type, spreading out from the bloodvessels and being inter- and intra-acinous in distribution (Gilbert and Chabrol). ^

Infarcts of the pyramids with lime salts occurred in 44 out of 48 cases specially examined (Goldschmid). ^

Thyroid. Fibrosis, epithelial hyperplasia, and colloid absorption, as in Graves' disease, have been described (Farrant).

Gilbert et Chabrol. *Arch. de méd. expér. et d'anat. path.*, Par., 1910, XXII, 896

Goldschmid. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1913, LVI, 77

Farrant. *Brit. med. Journ.*, 1914, i, 472.

Lando found some fibrosis, either inter- or intra-lobular, in every pancreas in 23 cases of cirrhosis. Steinhaus [1] found chronic interstitial pancreatitis in 11 out of 12 cases; Klippel and Lefas found it constantly in 8 cases of cirrhosis. I have seen extreme fibrosis and a calculus in the pancreas in fatal cirrhosis.

The pancreas is usually enlarged as a whole, but the tail is relatively more affected than the body and head of the gland. In his first series Lefas [2] found the weight increased by a third. Microscopically there is chronic interstitial pancreatitis, the fibrosis ~~being either perilobular, intralobular, or partly periacinous but chiefly intralobular~~. There are scattered areas of small-celled infiltration. Occasionally there is intralobular oedema. The gland cells undergo fatty and pigmentary change; but the islands of Langerhans remain intact. In the cirrhosis of haemochromatosis, however, there is such extensive interstitial pancreatitis, the islands of Langerhans being destroyed, that eventually diabetes (*diabète bronzé*) results. According to Klippel and Lefas there is no relation between the size and consistency of the pancreas and these changes in the liver, and in some instances the pancreas shews more advanced change than the liver. The change in the pancreas may be due to the same factors that set up cirrhosis of the liver (Klippel and Lefas), or in some cases it may be secondary to hepatic cirrhosis and the result of chronic venous engorgement (Lando [3]).

The *Kidneys*~~.—~~In the great majority of cases ~~of portal cirrhosis the kidneys~~ are free from ~~any~~ gross old-standing change, but are enlarged in about 50 per cent of the cases.

In 89 cases of fatal cirrhosis collected by Milian and Bassuet,[4] the kidneys were perfectly normal in 19; in 53, or 59·5 per cent, they were healthy or more or less hypertrophied. Pitt [5] found that when otherwise healthy the kidneys were enlarged in 50 per cent of the cases of cirrhosis.

~~This hypertrophy of the renal substance is a point of some interest as regards its explanation~~. In some instances the hypertrophy may merely be due to overwork from excessive drinking. It has been suggested that the hypertrophy is a compensatory process with the object of removing the toxic bodies which, owing to hepatic inadequacy, have flooded the circulation (Mollard [6]). In some instances enlarged kidneys are fatty or shew cloudy swelling. The kidneys are arteriosclerotic (granular) in about 25 per cent of cases dying with cirrhotic livers; and in about 5 per cent of the cases there is tubal nephritis. The relation of renal disease to cirrhosis is referred to again ~~(under the heading of associated morbid lesions, vide~~ on p. 224~~)~~.

Diaphragm.—In cases in which the abdomen has been distended for a considerable time, the diaphragm may shew marked muscular hypertrophy from overwork. It is, however, very seldom noticed.

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[1] Steinhaus. *Deutsch. Arch. f. klin. Med.*, 1902, lxxiv, 537.
[2] Lefas. *Arch. gén. de méd.*, Paris, 1900, clxxxv, 538.
[3] Lando. *Ztschr. f. Heilk.*, 1906 (*Abt. path. Anat.*), xxvii, 1.
[4] Milian et Bassuet. *Bull. Soc. anat.*, Paris, 1908, lxxviii, 337.
[5] Pitt. *Trans. Path. Soc.*, Lond., 1889, xl, 349.
[6] Mollard. *Lyon méd.*, 1902, xcix, 665.

There was great hypertrophy of the diaphragm in a patient who survived the Morison-Talma operation, for 2½ years.

Associated Lesions.—*Tuberculosis.*—Tuberculosis is met with in the bodies of patients with cirrhosis more often than in other non-tuberculous diseases.

Thus in 706 fatal cases of cirrhosis obtained by combining the statistics given by Lancereaux,[1] Pitt,[2] St. George's, Kelynack,[3] and Yeld,[4] 209, or 29·6 per cent, presented some evidence of tubercle. Taking the general incidence of tuberculosis in routine post-mortem work as 27·5 per cent (Osler[5]), and deducting the large number of cases dying directly from tuberculosis—about 14 per cent—it is evident that tubercle is more frequent in cirrhosis than in ordinary non-tuberculous diseases.

Alcohol and the other poisons which induce cirrhosis diminish the resistance of the body and so dispose to tuberculosis. Although a tuberculous cirrhosis (*vide* p. 346) has been described (Hanot and Gilbert[6]), there is no reason to think that ordinary cirrhosis is ever due to tuberculosis or to tuberculous peritonitis; when tuberculosis and cirrhosis are associated, the cirrhosis is, as a rule, the older condition.

Age plays some part in influencing the incidence of tuberculosis in patients with cirrhosis. Thus, while it is rare in the somewhat infrequent cases of hepatic cirrhosis in childhood, Pitt's statistics shew that alcoholic cirrhosis in patients under forty is accompanied by tuberculosis in two-thirds of the cases. The lungs and the peritoneum are the most frequent sites of tuberculosis in the subjects of cirrhosis. The tuberculosis may be old, acute, chronic or recrudescent, and not infrequently escapes detection during life.

Pulmonary Tuberculosis.—In patients with cirrhosis pulmonary tuberculosis is the direct cause of death in from 12 (Kelynack) to 14·5 per cent (Rolleston and Fenton). In cases of cirrhosis, whether fatal from the disease itself or from other diseases (excluding tuberculosis), the lungs are the most frequent site of tuberculous lesions.

In 584 cases of cirrhosis obtained by combining the statistics of Lancereaux, St. George's, Kelynack, and Yeld, tuberculous lesions were found in the lungs in 132, or 22·6 per cent. On the basis of Heitler's[7] estimate that tuberculosis is present in the lungs of 5 per cent of patients dying from diseases other than pulmonary tuberculosis—a low estimate—there is a marked increase in the incidence of tubercle in the lungs.

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[1] Lancereaux. *Traité des maladies du foie et du pancréas*, p. 302, 1899.

[2] Pitt. *Trans. Path. Soc.*, Lond., 1889, xl, 350.

[3] Kelynack. *Med. Chronicle*, 1897, vi, 262.

[4] Yeld. *St. Barth. Hosp. Rep.*, 1898, xxxiv, 215.

[5] Osler. *Practice of Medicine*, p. 295, 6th ed., 1905.

[6] Hanot et Gilbert. *Arch. gén. de méd.*, 1889, clxiv, 513.

[7] Quoted by Hilton Fagge. *Practice of Medicine*, vol. i, p. 953, 1886.

Pulmonary tuberculosis is more often found in patients who die from the effects of cirrhosis than in patients who have cirrhosis but die from other causes (excluding tuberculosis).

In 97 cases of cirrhosis in which death was not due to pulmonary tuberculosis tubercles were found in the lungs in 17. Of these 97 cases, 47 were fatal from the direct effects of cirrhosis, and pulmonary tuberculosis was found in 10, or 21·2 per cent; while in the remaining 50 cases in which cirrhosis was latent and death was due to some independent cause, pulmonary tuberculosis being of course excluded, tubercle was present in 7, or 14 per cent.[1]

Tuberculous Peritonitis.—Next to the lungs, the peritoneum is the most frequent situation of tuberculous infection in cirrhosis. In 584 cases of cirrhosis obtained by combining the statistics of Lancereaux, Fenton and myself, Kelynack, and Yeld there were 53 cases of peritoneal tubercle, or 9 per cent. In a large number of cases of peritoneal tubercle the lungs are previously infected. Tuberculous peritonitis is rare in adult males, and when it does occur is most often associated with hepatic cirrhosis. In 121 cases of cirrhosis Kelynack found active tuberculosis of the peritoneum in 12, in 4 of whom the peritoneum was alone affected. This suggests that cirrhosis of the liver disposes to the local invasion of the peritoneum by tubercle.

A certain degree of *simple chronic peritonitis* is often seen in the bodies of those who have died from cirrhosis of the liver, but a high degree of chronic peritonitis is comparatively rare in association with well-marked cirrhosis.

In Yeld's 131 cases of cirrhosis simple chronic peritonitis was present in 10·7 per cent, and in Sears and Lord's[2] 78 cases in 19 per cent.

The relation between cirrhosis and chronic peritonitis is probably not always the same; possibly in some cases the chronic peritonitis is secondary to the ascites induced by cirrhosis, the toxic bodies or bacteria in the ascitic fluid irritating the peritoneum; and it is conceivable that occasionally they are both related to alcoholism. In many instances the two processes are probably independent; thus, simple chronic peritonitis may be associated with and due to arteriosclerosis. As has just been pointed out, tuberculosis may supervene in the peritoneum.

Arteriosclerosis.—Since cirrhosis and arteriosclerosis occur about the same time of life, it is natural to find arterial degeneration of a greater or lesser degree in the bodies of patients dying from cirrhosis. There is, however, no direct relation between these two conditions; cirrhosis is accompanied by a low blood-pressure, and thus does not tend to set up arterial degeneration, while endarteritis is not a cause of genuine hepatic cirrhosis. In some exceptional instances syphilitic endarteritis is associated with hepatic cirrhosis (probably parasyphilitic) in children, and in the rare disease haemochromatosis, cirrhosis of the liver and endarteritis of the hepatic artery may occur. From the statistical point of view, arteriosclerosis is the commonest, and one of the least essential, morbid changes found in the bodies of patients with hepatic cirrhosis.

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[1] Rolleston and Fenton. *Birmingham Med. Rev.*, 1896, xl, 193.
[2] Sears and Lord. *Boston Med. and Surg. Journ.*, 1902, cxlvii, 285.

In 78 cases of cirrhosis tabulated by Sears and Lord, arteriosclerosis was noted in 49 instances; often, however, there was only a small patch in the aorta.

The *heart* is frequently flabby and dilated, and the muscular tissue often shews cloudy swelling or fatty degeneration, due to toxaemia or alcoholism. In uncomplicated cirrhosis the heart may be smaller than natural (Carnot[1]); this is associated with the habitually low blood-pressure in cirrhosis.

In 114 cases of cirrhosis, obtained by uniting Cheadle's[2] and Sears and Lord's statistics, there was fatty degeneration in 33, or 28·9 per cent, and myocarditis in 3.

In rare instances infective endocarditis may occur as a complication. Chronic valvulitis, like arteriosclerosis, is frequently associated with cirrhosis. Although adherent pericardium does not induce genuine hepatic cirrhosis, calcification of the pericardium is said to be associated in the majority of cases with definite cirrhosis (*vide* p. 98).

Renal Disease.—The kidneys in fatal cases of cirrhosis are, generally speaking, free from any marked structural change; they often shew enlargement from overwork (drinking), chronic venous engorgement from cardiac failure, or some degree of tubal changes, such as fatty degeneration or cloudy swelling, from toxaemia or alcohol. When definite structural change is present, it is usual to find that the kidney is granular from the effects of arteriosclerosis; definite lardaceous disease and tubal nephritis are quite rare. The association of granular kidney with hepatic cirrhosis is intelligibly explained by the fact that the two diseases are common at the same time of life, and are therefore met with together in a certain number of cases. But though both the liver and kidney are very liable to fibrosis, this change in the two organs respectively is due to different morbid agencies (Dickinson[3]).

In an analysis of 78 fatal cases of cirrhosis, Sears and Lord[4] found chronic nephritis (interstitial or tubular) in 23, or 29·4 per cent, fatty change in 15, and lardaceous change in 2. In 440 cases of cirrhosis of the liver obtained by combining the statistics of Pitt,[5] Kelynack,[6] Yeld,[7] Cheadle, and myself[8] there were 110 with distinct fibrosis of the kidneys, or 25 per cent. In some of the cases the renal change was not very marked, and as the average age for death

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[1] Carnot. *Progrès méd.*, Paris, 1909, v, 61.
[2] Cheadle, W. B. *Some Cirrhoses of the Liver*, Lond., 1900, p. 47.
[3] Dickinson, W. H. *Med.-Chir. Trans.*, 1873, lvi, 34.
[4] Sears and Lord. *Boston Med. and Surg. Journ.*, 1902, cxlvii, 285.
[5] Pitt, G. N. *Trans. Path. Soc.*, Lond., 1889, xl, 348.
[6] Kelynack, T. N. *Birmingham Med. Rev.*, 1897, xli, 86.
[7] Yeld, R. A. *St. Barth. Hosp. Rep.*, 1898, xxxiv, 215.
[8] Rolleston and Fenton. *Birmingham Med. Rev.*, 1896, xl, 193.

in cirrhosis is over forty-five years, it is probable that in some instances the change in the kidney was senile. Dickinson estimated that well-marked granular kidneys occurred in 15 per cent of the cases of cirrhosis.

As would be naturally expected from the greater frequency of both hepatic cirrhosis and arteriosclerosis in men, the two conditions are more often seen associated in the male than in the female sex.

The question whether a granular kidney is more often associated with a large or with a small cirrhotic liver has been referred to already (p. 197). Pitt's and my own observations point to a small cirrhotic liver as more frequently associated with a granular kidney; Price's statistics were to the opposite effect.

Cirrhosis complicated with Carcinoma, etc.—The form of primary carcinoma of the liver described by Hanot and Gilbert as cancer with cirrhosis is essentially cirrhosis first with a secondary hyperplasia of the liver cells. At first this hyperplasia is compensatory and gives rise to cirrhosis with adenoma; subsequently the cellular proliferation becomes so riotous as to be malignant and constitutes carcinoma. It is very hard to draw the line between the different stages of cirrhosis, cirrhosis with adenoma, and carcinoma with cirrhosis (*vide* p. 474).

Secondary Carcinoma in a Cirrhotic Liver.—Cirrhosis of the liver and carcinoma elsewhere in the body are both so common that it is remarkable that secondary growths are so rare in cirrhotic livers. In a case in St. George's Hospital there was a secondary growth in a cirrhotic liver, the primary growth being a carcinoma of the pylorus. In 608 cases of secondary malignant disease of the liver the organ was cirrhotic in two (Colwell[1]). Hale White[2] refers to a case of sarcoma of bone with a secondary growth in a cirrhotic liver, and Poulain[3] and Achard and Laubray[4] to cases secondary to carcinoma of the colon and stomach. The coexistence of a cirrhotic liver (without any secondary growths) and malignant disease in some other part of the body is also rare; in 2634 necropsies at the Middlesex Hospital on cases of malignant disease hepatic cirrhosis was present in 27 only (Colwell).

Other Accidental Lesions in the Liver.—In a few cases a hydatid cyst has been found embedded in a cirrhotic liver (*vide* p. 397). Among 174 cases of hepatic gumma, obtained by combining J. L. Allen and Flexner's[5] cases, universal cirrhosis was present in 13, or 7·5 per cent. Tubercles are sometimes implanted in a cirrhotic liver. It is rare to find cirrhosis and lardaceous disease combined. A tight-laced liver may be cirrhotic (*vide* Fig. 6).

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[1] Colwell. *Arch. Middlesex Hosp.*, 1905, v, 135.
[2] Hale White. Allbutt's *System*, 1897, iv, 208.
[3] Poulain. *Bull. Soc. anat.*, Paris, 1899, 6. s., i, 1089.
[4] Achard et Laubray. *Bull. et mém. Soc. méd. des hôp. de Paris*, 1902, 3. s., xix, 335.
[5] Flexner. *New York Med. Journ.*, 1902, lxxv, 101.

Clinical Features

The course of ordinary cirrhosis may be divided into: (1) The early or pre-ascitic stage, which is often divided into two by early haematemesis. (2) The late or ascitic stage.

Cases may die from intercurrent diseases during the course of cirrhosis or may be cut off by a very profuse gastro-intestinal haemorrhage long before the onset of ascites. Again, complications, such as tuberculosis, cardiac failure, peripheral neuritis, or other manifestations of alcoholism, may supervene and prove fatal or mask the symptoms of cirrhosis.

The *early stage* begins gradually and vaguely; though in some cases it may be dated back to some acute illness or to a passing attack of gastro-enteritis. The symptoms are mainly those of dyspepsia, often of an alcoholic nature, with loss of appetite, occasional nausea and vomiting, especially in the morning, flatulence, abdominal pain or unrest, and looseness or irregularity of the bowels. Sometimes symptoms are absent; at other times they are those of chronic alcoholism. Bodily strength, vigour, and energy may be seriously impaired, and the condition resembles neurasthenia; or, on the other hand, there may be little or no complaint of altered health.

There may be some heaviness or tenderness in the epigastrium and hypochondrium, especially after meals, which is partly due to dyspepsia and perhaps partly to hepatic enlargement. The liver is generally somewhat enlarged and may be slightly tender. The enlargement may vary considerably within short intervals, and is perhaps due to engorgement. The spleen is usually not enlarged, but it may be palpably so, especially shortly before haematemesis supervenes.

Epistaxis may occur now and then, but the most important event in the early stage is the occurrence of haematemesis. This may come on after some premonitory discomfort and fever, or may occur with little or no warning, except perhaps slight faintness immediately before a large quantity of blood is brought up. It is very rarely fatal, and is very seldom repeated within a short interval. After it the patient is blanched and anaemic for a time, but soon recovers.

Usually some months or even years elapse after haematemesis before ascites develops, especially if the patient has taken warning and altered his manner of life. The disease may then become latent from the development of compensatory processes. But in cases of acute cirrhosis, in which there is fever, the effects of haematemesis are hardly recovered from before ascites begins.

The Late or Ascitic Stage.—The onset of ascites is somewhat sudden; when it occurs quite suddenly and with great rapidity it may be due to another factor, viz. thrombosis of the portal vein. The onset of ascites may be preceded by gaseous distension of the intestines, which for a time

may mask the ascites. Oedema of the feet may also precede instead of follow the appearance of ascites.

When ascites occurs, the patient has undergone a considerable amount of wasting, shews marked muscular debility, and is unfit to be out of bed. The wasting of the temporal and facial regions gives a characteristic appearance to the patient. The atrophy of the thoracic muscles shews up the bony skeleton, and contrasts with the swollen abdomen. The skin becomes dry, harsh, and loses its elasticity, and may shew petechiae, while there may be haemorrhages from the various mucous membranes and oozing from the gums. The ascites may require tapping once, twice, or even oftener, but in cases of cirrhosis uncomplicated by chronic peritonitis paracentesis is seldom required more than twice. The patient emaciates rapidly, loses strength, becomes stupid, drowsy, or even delirious, and dies, sinking down in the bed without any great reaccumulation of fluid in the peritoneal cavity. A patient often goes downhill very rapidly after paracentesis, not because of the tapping, but from the advanced stage of the disease. Sometimes, in fact, ascites disappears, while the patient becomes more drowsy and uraemic, and passes into what may be spoken of as a post-ascitic stage. The patient may then linger on in a semi-comatose condition for some weeks, and die from an acute and terminal infection or gradually from mere weakness.

Latency.—Cirrhosis of the liver is not uncommonly latent and gives rise to no symptoms. It may be found in the bodies of persons who have died as the result of accidents or from other diseases. Of 82 cases in which cirrhosis was found after death at Charing Cross Hospital, 12 were in fatal street accidents (Mott and Candler[1]). It would be incorrect to say that when a patient dies from some other disease the cirrhosis has necessarily been entirely latent, since patients with cirrhosis rapidly die with pulmonary tuberculosis and are very bad subjects for pneumonia, erysipelas, and other acute infections. The fact, however, that out of 167 consecutive necropsies at St. George's Hospital in which the liver was cirrhotic, 86, or just over half, died from other diseases and not directly from cirrhosis, shews how frequently cirrhosis remains latent. It thus differs from universal chronic perihepatitis, which is practically always accompanied by ascites. The latency of portal cirrhosis depends on the compensatory processes, *i.e.* the collateral circulation between the branches of the portal and general systemic veins, and hyperplasia of the liver cells. These two compensatory mechanisms are further discussed on p. 291.

When cirrhosis becomes latent, the liver is enlarged as the result of compensatory hyperplasia of the liver cells. Hanot and Gilbert[2] insisted on the enlarged liver of latent cirrhosis, and spoke of it under the title of hypertrophic alcoholic cirrhosis. Cirrhosis is more often latent in men than in women. This is not merely because cirrhosis is commoner in men than in women, for the ratio of women to men is lower among the cases dying independently of cirrhosis than among the cases fatal from the direct effects of cirrhosis.

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[1] Mott and Candler. *Arch. Neur. Path. Lab., Lond. County Asyl., Claybury*, 1907, iii, 139.

[2] Hanot et Gilbert. *Bull. et mém. Soc. méd. des hôp. de Paris*, 1890, 3. s., vii, 493.

In 167 cases in which the liver was cirrhotic at the necropsy at St. George's Hospital, 121 were males and 46 females, or a ratio of 5 to 2. Of these, 80 died from the direct effects of cirrhosis, 50 being males and 30 females, a ratio of 5 to 3; while of the 87 who died from other factors independent of cirrhosis, 71 were males and 16 females, a ratio of 4½ to 1.

Minor Signs and Symptoms.—*Facial Aspect.*—The face may be bloated and shew rosacea due to dyspepsia, either induced by, or independent of, alcoholism; the area of skin affected involves the nose and the cheeks and roughly corresponds with the common site of lupus erythematosus, the so-called "flush area." Galloway[1] recorded a case with subsequent atrophy of the skin. The condition of the skin of the face varies; in early cases it may be pale and clear, but often it is sallow, muddy, and dirty-looking. This pigmentation is sometimes so accentuated as to suggest that the cirrhosis may be a terminal stage of haemochromatosis. In the more advanced stages the face is drawn and thin, ~~the eyes deeply set~~, and the conjunctivae congested and muddy, or slightly icteric. The wasting in the temporal regions is often very manifest. The skin of the face may present clusters of dilated vessels or stigmata; these may be capillary or in some instances arterial, as shewn by pulsation and by the character of their bleeding, which may occur on slight provocation. It is an interesting question why these stigmata should develop in cirrhosis; they cannot be due to increased pressure in the portal system. It has been thought that they depend on alcoholism; Bouchard[2] has suggested that, owing to altered internal secretion of the liver, the arteries all over the body undergo a special change resulting in local dilatations. The lips are usually dry and apt to be fissured; the tongue flabby, furred, or dry, and sometimes tremulous. The gums are apt to become spongy and, when hepatic insufficiency has become established, to bleed. The throat is often chronically congested, and pharyngitis and laryngitis are frequent.

The *skin* of the body is often dry and harsh with loss of elasticity. Probably from failure of the antitoxic function of the liver eruptions such as erythema exudativum may occur. Angioneurotic oedema has been observed (Corner[3]). Local haemorrhages may ~~occur as the result of~~ follow slight or unnoticed injuries ~~traumatism~~. When cirrhosis is advancing, small angiomas may crop up all over the surface of the body, and in exceptional instances may unite to form areas of considerable extent. A remarkable case of multiple haemangiomas, many of which were solid, has been recorded by Winternitz and Boggs.[4] Bouchard records a case

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[1] Galloway. *Brit. Med. Journ.*, 1908, i, 665.

[2] Bouchard. *Rev. de méd.*, 1902, xxii, 837.

[3] Corner. *Lancet*, Lond., 1909, ii, 295.

[4] Winternitz and Boggs. *Johns Hopkins Hosp. Bull.*, 1910, xxi, 203.

^ Exophthalmos, unilateral or bilateral was found in 10 out of 12 cases specially examined (Farrant).

Farrant. *Brit. med. Journ.*, 1914, i, 475.

Haemolytic jaundice may
supervene (Chalier)

and may then be associated
with the onset of ascites
and haemorrhage: (Fiessinger
and Brodin's acute ictero-ascitic
syndrome.

Chalier. *Rev. de méd.*, Par., 1911 (Jubilé du Prof. Lepine)
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Fiessinger et Brodin. *Presse méd.*, Paris, 1924, 12 in which angiomas appeared when the disease was advancing and receded when improvement took place. According to Gilbert and Herscher,[1] capillary angiomas are commoner on the trunk, and arterial on the hands and face. A zone of dilated capillaries on the chest about the attachment of the diaphragm has no real significance, since it is seen in many men of middle age, especially those with emphysema, who have nothing wrong with their livers.[2] Dilatation of the subcutaneous veins around the umbilicus (cirsomphalos) points to portal obstruction; when well marked, this is spoken of as a "caput Medusae." Trousseau[3] described a palpable thrill as well as a bruit in these veins. The collateral circulation around the umbilicus, due to portal obstruction, must be distinguished from dilatation and varicosity of the superior and inferior epigastric veins in obstruction of the inferior vena cava. This latter anastomosis is often developed in a minor degree as the result of ascites pressing on the inferior vena cava. In pure portal obstruction the visible dilated veins are mainly around and above the umbilicus; the dilated veins due to obstruction of the inferior vena cava are first seen below the umbilicus (*vide* p. 249).

Jaundice is not a prominent feature in portal cirrhosis, but it is met with at some time in the course of the disease, as shewn by statistics, in more than one-third of the cases.

Thus, in 293 cases, obtained by combining the statistics of Fagge,[4] Yeld,[5] and Sears and Lord,[6] jaundice was recorded in 107, or 36·5 per cent.

The jaundice is usually slight and often transient. It may be merely an incident in the course of the disease, and have passed away before the patient comes under observation with indubitable cirrhosis. Not uncommonly there is slight jaundice with staining of the skin and blood serum, but no bile pigment in the urine, or "acholuric jaundice" (Gilbert and Herscher[7]). ⋏

Definite, well-marked jaundice may be catarrhal and due to gastro-duodenal inflammation — an exceedingly common event in alcoholic subjects. It is not infrequent in cases of acute cirrhosis, and is then probably due to inflammation of the small intrahepatic bile-ducts. In rare instances it is dependent on a gall-stone in the common duct, or terminal and the result of acute and widespread degenerative changes in the liver cells (icterus gravis)⋏

Clubbing of the fingers, also spoken of as "Hippocratic fingers," which is sometimes seen in long-standing cases of hypertrophic biliary cirrhosis, is extremely rare in portal cirrhosis.

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[1] Gilbert et Herscher. *Compt. rend. Soc. Biol.*, 1903, lv, 167.

[2] For further information about this "costal fringe" see Solis-Cohen (*Am. Journ. Med. Sc.*, 1894, cviii, 135), F. P. Weber (*Edin. Med. Journ.*, 1904, N.S., xv, 346), and Haeberlin (*Deutsch. Arch. f. klin. Med.*, Leipz., 1908, xciii, 43).

[3] Trousseau. *Lectures on Clinical Medicine*, v, 131, Transl. New Syd. Soc., 1872.

[4] C. Hilton Fagge and Pye-Smith. *Practice of Medicine*, ii, 270, 1886.

[5] Yeld. *St. Barth. Hosp. Rep.*, 1898, xxxiv, 215.

[6] Sears and Lord. *Boston Med. and Surg. Journ.*, 1902, cxlvii, 285.

[7] Gilbert et Herscher. *Presse méd.*, Paris, 1903, p. 541.

Flückiger[1] in 1884 and Bouchard[2] in 1890 described cases. I watched it develop in a patient in whom, two and a half years before, Morison's operation for the relief of ascites had been successfully performed. There was no other obvious cause, such as pulmonary or heart disease. Obermayer[3] reported a case of portal cirrhosis with hypertrophic osteo-arthropathy.

Debility.—Marked weakness and languor out of proportion to the physical signs are not uncommon, and may be the first symptoms calling for treatment, before there are sufficient data for diagnosing cirrhosis. Muscular weakness and asthenia are usually constant in the late stages of the disease, and are only natural when the poor state of general nutrition is taken into account. The debility is partly due to the toxaemia of hepatic inadequacy.

Wasting in advanced cirrhosis is usually very considerable; the muscles of the trunk, extremities, and face become flabby and atrophied. The limbs become like spindles. The face gets sharpened, the fat disappears from the buccal pads, and the temporal fossae fall in, so that the face often has the aspect of a case of advanced pulmonary tuberculosis or of malignant disease. In latent cirrhosis, however, bodily nutrition may be very good, and there may be a thick layer of fat over the body.

I have several times seen somewhat fat patients with latent hepatic cirrhosis die from rapid or generalised tuberculosis. In such cases the existence of cirrhosis is very likely to be overlooked. The large size of the liver is, however, fairly constant.

Offensive Breath.—In some, but certainly not in all, patients with cirrhosis the breath is very offensive without any local cause, such as decayed teeth, chronic follicular tonsillitis, or ozaena. The odour is often peculiar, and is described as cadaveric or earthy. It resembles the smell of dried and decomposing blood, and is sometimes associated with oozing from the gums or epistaxis. It is probably due to failure of the antitoxic function of the liver and to the passage into the general circulation of poisons manufactured in the alimentary canal. In other words it depends on hepatic inadequacy, and is therefore of bad omen.

Experimentally Roger and Garnier[4] find that in healthy rabbits a hypodermic injection of sulphuretted hydrogen is followed by excretion of the gas by the lungs, but that about double the quantity must be injected into the rectum before it passes through the lungs. When the functional activity of the rabbit's liver has been reduced by the subcutaneous injection of phosphorated oil, a much smaller rectal injection of sulphuretted hydrogen was followed by the excretion of the gas by the lungs.

The amylolytic power of the *saliva* is not necessarily reduced; it may even be increased (Robertson[5]). If there is ascites or diarrhoea, the amount of saliva will tend to be diminished.

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[1] Flückiger. *Wien. med. Wchnschr.*, 1884, xxxiv, 1457.

[2] Bouchard. Quoted by Klippel et Vigouroux. *Presse méd.*, 1903, i, 245.

[3] Obermayer. *Wien. klin. Rundschau*, 1897, xi, 625.

[4] Roger et Garnier. *Compt. rend. Soc. Biol.*, Paris, 1898, l, 714.

[5] Robertson, W. G. A. *Journ. Path. and Bacteriol.*, 1901, vii, 118.

Cases have also been described by
Winternitz and Boggs

Winternitz and Boggs Johns Hopkins Hosp. Bull., Balt., 1910, XXI, 203

V. Leube[1] mentions a case of excessive salivation in which 3 litres were secreted in one twenty-four hours, and as a result ascites, which was present, disappeared. Cirrhosis of the liver was found after death.

Dyspepsia is almost constant in portal cirrhosis, and usually precedes, often for some years, the first striking manifestation of the disease, namely, haematemesis. It may be irritative and intermittent, or flatulent and very persistent. In the early stages it is due to gastritis—often of alcoholic origin—and has a causal relation to dyspeptic cirrhosis, inasmuch as the chronic gastric catarrh manufactures the toxins which are carried to the liver and induce cirrhosis. Later, when portal obstruction and engorgement of the stomach and intestines have supervened, gastritis and indigestion are aggravated. The portal obstruction favours infection and at the same time interferes with the digestion and absorption of food. Absorption is also impaired as the result of the curtailed length of the intestinal tract; the process of digestion is interfered with from a deficiency in the secretion of hydrochloric acid. Exceptionally hyperchlorhydria is present. Hewes[2] has recorded two cases of gastrosuccorrhoea associated with cirrhosis. There is loss of appetite for solid food, especially for meat. Morning vomiting may be present, and is naturally more often seen in alcoholic subjects. Occasionally acute or subacute attacks of gastritis may supervene, and if seen for the first time in one of these exacerbations, the underlying condition of cirrhosis may not at first be manifest.

Tympanites may come on acutely or subacutely in the late stage of cirrhosis. It is probably toxic (Wilkinson[3]).

Diarrhoea.—Comparatively early in the disease diarrhoea or excessive looseness of the bowels may be due to a general catarrh of the intestinal tract in alcoholic subjects, and to diminished and delayed absorption. In other cases diarrhoea may alternate with the more usual state of constipation. Diarrhoea is more often met with in multilobular cirrhosis in children.

In the last stage of the disease, when the patient is in a toxaemic state, diarrhoea may set in and be very rebellious. It may continue until the patient dies of exhaustion. It is, however, possible that the diarrhoea is, like that of renal disease, of use in getting rid of toxic substances.

Constipation is often present. Chronic catarrh of a certain degree may cause it instead of diarrhoea, by impairing the peristaltic powers of the intestine; chronic portal engorgement impairs both the muscular and nervous activity of the bowel. In the later stages ascites helps to produce constipation, both by weakening the muscular walls of the abdomen and by withdrawing water from the bowel. Thus a patient with ascites may chiefly complain of constipation. Moreover, milk diet leaves little residue for the faeces and may thus accentuate this tendency. Constipation leads to putrefaction and fermentation and so to auto-intoxication.

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[1] v. Leube. *Deutsches Arch. f. klin. Med.*, 1899, lxvi.
[2] Hewes. *Boston Med. and Surg. Journ.*, 1906, clv, 163.
[3] Wilkinson. *Med. Chron.*, Manchester, 1910, li, 212.

The *urine* is usually diminished in amount in the late stages of cirrhosis. The smaller quantity is closely associated with, in fact mainly depends on, low arterial pressure, which in turn is correlated with increased blood-pressure in the portal vein. Normally more water passes through the kidneys during digestion, owing to increased absorption from the alimentary canal; in cirrhosis, absorption is delayed on account of the increased pressure in the portal vein, and, as a result, the normal relation between the amount of urine excreted during digestion and in the intervals is reversed, more urine being excreted during fasting. This phenomenon, described by Gilbert and Lereboullet,[1] has been termed "opsiuria."

Another abnormal feature in the excretion of urine in cirrhosis, described by Chauffard and Castaigne,[2] is that methylene-blue when taken by the mouth is excreted intermittently, and not continuously as in health.

The urine is strongly acid, usually of a high specific gravity, high coloured, often reddish orange, from the presence of uroerythrin, and, like the urine of cardiac disease, commonly precipitates urates on standing.

The amount of urobilin is increased in cirrhosis,[3] while the pigment described as urohaematoporphyrin, which is in reality not a definite chemical compound, but a mixture of a large quantity of haematoporphyrin with a small quantity of urobilin, has been said to occur in the urine of some cases of cirrhosis (McMunn[4]). Indican may also appear in the urine without any special evidence of intestinal disturbance.

Since jaundice is somewhat uncommon in portal cirrhosis, bile pigment is not often found in the urine, but it may be present without any manifest staining of the skin. ~~I have seen this during the comatose condition preceding death.~~ ~~The converse is more often seen, viz~~. slight icteric coloration of the skin, due to small quantities of bile pigment in the blood serum, without any bile pigment in the urine (acholuric
is sometimes seen
jaundice) t~~hough the amount of~~ urobilin in the urine is increased, Gilbert and Herscher[5] believe that bilirubin is transformed by the kidneys into urobilin; this condition was formerly called "urobilin jaundice" (*vide* p. 534). Strauss[6] found the fatty acids increased.

In cases of advanced cirrhosis a port-wine coloration is, in rare instances, obtained when liquor ferri perchloridi is added to the urine; in the absence of drugs, such as salicylates, aspirin, salol, diuretin, which give a similar colour reaction, this is due to the presence of diacetic acid (Gerhardt's reaction).

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[1] Gilbert et Lereboullet. *Compt. rend. Soc. Biol.*, Paris, 1901, liii, 276.

[2] Chauffard et Castaigne. *Ibid.*, 1898, l, 359.

[3] Garrod, *Lancet*, 1900, ii, 1323 (Bradshaw lecture); Durandeau, *Thèse de Paris*, 1900, No. 346.

[4] McMunn. *Journ. Physiol.*, 1889, x, 71.

[5] Gilbert et Herscher. *Presse méd.*, Dec. 27, 1902; 1903, p. 541.

[6] Strauss. *Deutsche med. Wchnschr.*, 1901, xxvii, 759.

Dissociated bilirubinuria without bile salts in the urine or more often the reverse, may occur.

and is often combined with bile salts but no pigment in the urine;

If the urine is previously heated, the coloration is less marked; this is due to the transformation of diacetic acid into acetone, which does not give this coloration with ferric chloride. The presence of diacetic acid points to the presence in the blood of organic acids, such as β-oxybutyric acid, or to acidosis. This condition is one of great gravity, inasmuch as it may pass into coma. When the urine gives the reaction for diacetic acid, bicarbonate of sodium should be given in large doses, and if coma begins to appear, transfusion of saline solution with bicarbonate of sodium (2 drams to the pint) should be performed. As is well known, starvation, especially in women with gastric ulcer, may induce diaceturia without any other sign of acid intoxication.[1]

When the disease is well established, the amount of *urea* excreted is diminished, while that of uric acid is increased. The diminution in the output of urea accompanies, but is not a direct result of, destruction of the liver cells; it is correlated with an increase in the nitrogen excreted in the form of ammonia in the urine. The nitrogen in the form of ammonia may be present in the urine in the normal amount (2 to 5 per cent of the total nitrogen), but when destructive changes in the liver cells are active it may rise to as much as 20 per cent of the total nitrogen. This increase in ammonia is correlated with a diminished formation of urea. According to Herter,[2] the increase in ammonia is not due to inability of the liver cells to form urea—for, as shewn by Weintraud, if ammonia is given to such patients it appears in the urine as urea—but to the fact that the ammonia is seized upon by organic acids before it can be transformed into urea. The ammonia neutralises the organic acid and so tends to prevent acidosis. In advanced cases with extensive destruction of the liver cells leucine and tyrosine may be found in the urine.

The *chlorides* are diminished when there is ascites.

Albuminuria is comparatively rare. When it does occur, it is usually due to some concomitant organic renal disease, such as granular kidney, tubal nephritis, or lardaceous change. In 89 cases tabulated by Milian and Bassuet[3] albuminuria occurred in three.

As a result of hepatic insufficiency toxaemia commonly occurs in the late stages of cirrhosis, and sometimes so affects the renal tubules as to give rise to albuminuria and casts. Albuminuria is sometimes associated with cardiac failure, and probably toxaemia combined with chronic venous engorgement of the kidney is the most favourable condition for the production of albuminuria. In rare cases albuminuria may be mechanical and due to the pressure of a considerably enlarged spleen on the left renal vein and possibly on the left kidney. Falkenheim[4] in 1884, and in 1902 the writer,[5] observed intermittent albuminuria due to this cause in cirrhosis. In these cases there is considerable albuminuria with high-coloured and uratic urine, thus resembling that of chronic venous engorgement, when the patient lies on the left side or on his back; when the patient is in the erect position, lies on the right side or on his face, the urine is free from albumin (Falkenheim).

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[1] *Vide* Rolleston and Tebbs. *Brit. Med. Journ.*, 1904, ii, 114.
[2] Herter, C. A. *Lectures on Chemical Pathology*, p. 347, 1902.
[3] Milian et Bassuet. *Bull. Soc. anat.*, Paris, 1903, lxxviii, 337.
[4] Falkenheim. *Deutsch. Arch. f. klin. Med.*, 1884, xxxv, 446.
[5] Rolleston. *Lancet*, 1902, i, 585.

Albumose is very occasionally observed in the urine when hepatic inadequacy has supervened, and may be associated with albuminuria (Teissier[1]).

Haematuria is distinctly rare; it may be due to concomitant subacute nephritis in a previously damaged kidney, or to toxaemia and occur at the same time as epistaxis and oozing from the gums.

Glycosuria is rare in ordinary cirrhosis. Glycosuria occurs in a high proportion of the cases of pigmented cirrhosis from haemochromatosis (*diabète bronzé*; *vide* p. 303), but it is then due to a concomitant fibrosis of a very intimate nature in the pancreas—a change which is not present in ordinary cirrhosis. The great rarity of glycosuria in cirrhosis is certainly curious, since the liver is usually regarded as stopping sugar reaching it from the alimentary canal. It might naturally be expected that sugar would pass through the cirrhotic liver, or that by means of the compensatory venous anastomoses some sugar would avoid the liver and pass into the general circulation, and so appear in the urine. Brault's[2] histological examinations of cirrhotic livers shew that the glycogenic function of the liver is well maintained, and agree with the great rarity of glycosuria. It has sometimes been observed that if an excessive amount of sugar in the form of syrup is given to a patient with cirrhosis, alimentary glycosuria is produced when it would not occur in a healthy person.

In a normal person 3½ ounces (100 grams) of sugar given in half a pint of water or tea on an empty stomach will not produce alimentary glycosuria. To test for alimentary glycosuria this quantity of sugar should be given before food and the urine should be obtained after six hours and tested for sugar (Strauss[3]).

Alimentary glycosuria is by no means constant in cirrhosis; in 95 collected cases it was positive in 42 (Simon[4]); even if it were more frequent it would not prove that the liver was at fault, for it appears that it is to be correlated with changes in the pancreas (Steinhaus[5]). According to most observers, such as Kraus and Ludwig,[6] Bloch,[7] Colasanti,[8] Linoissier and Roque,[9] Gorget,[10] Ingelnans and Dehon,[11] and

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[1] Teissier. *Semaine méd.*, Paris, 1899, xix, 282.
[2] Brault. *Arch. de méd. expér. et d'anat. path.*, 1902, xiv, 453.
[3] Strauss. *Deutsche med. Wchnschr.*, 1901, xxvii, 757.
[4] Simon. *Clinical Diagnosis*, p. 569, 1911.
[5] Steinhaus. *Deutsch. Arch. f. klin. Med.*, 1902, lxxiv, 537.
[6] Kraus und Ludwig. *Wien. klin. Wchnschr.*, 1891, iv, 855, 897.
[7] Bloch. *Ztschr. f. klin. Med.*, 1893, xxii, 525.
[8] Colasanti. *Arch. ital. de biol.*, 1892, xvii, 335.
[9] Linoissier et Roque. *Arch. de méd. expér. et d'anat. path.*, 1895, vii, 228.
[10] Gorget. *Rev. de méd.*, 1897, xvii, 537.
[11] Ingelnans et Dehon. *Arch. de méd. expér. et d'anat. path.*, 1903, xv, 189.

Churchman,[1] alimentary glycosuria is a most unreliable sign of hepatic insufficiency. Alimentary laevulosuria, however, has been thought to be of some value as an indication of hepatic inadequacy (Naunyn,[2] Halász[3]), and is said to be almost constant in cirrhosis (Goodman[4]). The glycogenic function may be retained while other functions have failed, as is commonly seen in cirrhosis; again, sugar taken into the alimentary canal may be delayed in absorption and undergo fermentation, thus vitiating the test. Cammidge[5] obtained a positive pancreatic reaction in 14 out of 21 cases. Chyluria has been observed in exceptional cases.[6]

Urotoxic Coefficient.—In cases with hepatic insufficiency, due to destructive changes in the liver cells, the toxicity of the urine has been found to be increased (Surmont,[7] Gorget), as shewn by the fact that when injected into dogs it appeared to be decidedly more toxic than healthy urine (Roger[8]).

Symptoms connected with the Vascular System.—From a flabby, possibly fatty, condition of the myocardium the left ventricle may be dilated and a systolic apex murmur and accentuated second sound over the pulmonary artery, due to mitral regurgitation, may be present. When the heart is displaced upwards by ascites, the apex-beat may be in the left fourth or even in the third intercostal space, and from slight kinking of the pulmonary artery a systolic murmur may be produced in that area. The murmurs at the apex and over the pulmonary artery may disappear after paracentesis of the abdomen. The impulse is, as a rule, feeble. The second sound over the aorta is rather less distinct than in health, and is correlated with the low arterial blood-pressure.

Fatty degeneration of the heart may account for sudden death in cases of cirrhosis.

The following is a good example of gradual cardiac failure occurring in the course of cirrhosis.

A man aged fifty-six who died under my care in St. George's Hospital had general oedema, slight jaundice, albuminuria, a low-tension pulse, and wandering delirium. He had in previous years had jaundice and haematemesis. His respirations were very rapid (56); he had signs of a small pleural effusion on the right side and haemoptysis. The necropsy shewed marked multilobular cirrhosis, kidneys free from old disease, right pleural effusion, collapse of lower lobe of right lung, but no pulmonary apoplexy or tubercle. The heart, 21 ounces, was dilated and hypertrophied, and shewed fatty degeneration in both ventricles. The valves were healthy.

The pulse-rate is usually slightly quickened; in the late stages when there is general toxaemia the pulse may be rapid and in coma there may be tachycardia. The blood-pressure is ~~rather~~ low when the disease is active or producing symptoms and ~~falls~~ after paracentesis, but when it is latent or compensated for it may be normal. According to Gilbert and his pupils,[1] the low blood-pressure is a mechanical effect due to the increased portal engorgement. Herrick's[2] view that in portal cirrhosis there is a freer communication between the hepatic artery and the portal vein, which accounts for the increased pressure in the portal vein, would also to some extent explain the fall of arterial pressure. It would, however, appear highly probable that the low tension is a toxic effect. The low blood-pressure is associated with an increased rate of the pulse and with a small output of urine. Capillary pulsation with healthy aortic valves has been observed (Bouchard[3]); and pulsation of the veins of the forearm (Hitschmann[4]) has been recorded.

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[1] Churchman. *Johns Hopkins Hosp. Bull.*, 1912, xxiii, 10.
[2] Naunyn. *Diabetes Mellitus*, 1900.
[3] Halász. *Wien. klin. Wchnschr.*, 1908, xxi, 44.
[4] Goodman. *Journ. Am. Med. Assoc.*, Chicago, 1909, liii, 2054.
[5] Cammidge. *Proc. Roy. Soc. Med.*, Lond., 1910, iii (Med. Sect.), 174.
[6] *Boston City Hosp. Rep.*, U.S.A., 1911, 27.
[7] Surmont. *Arch. gén. de méd.*, 1892, i, 162.
[8] Roger. *Presse méd.*, 1897, i, 293.

The Blood.—In early or latent cases when the general health is well maintained the blood is practically normal. When definite symptoms appear there is usually a secondary anaemia which becomes more marked as the disease progresses. In Emerson's[5] 32 cases the red blood-corpuscles varied from 3,100,000 to 5,000,000, with a mean of 4,500,000; the average haemoglobin percentage was 68. From repeated haemorrhages a grave secondary anaemia with normoblasts may be produced. Occasionally a grave anaemia develops in patients with cirrhosis without any manifest cause, such as haemorrhage.

In a case quoted by Cabot[6] the blood count was as low as 1,300,000 reds and 22 per cent of haemoglobin. Von Limbeck[7] refers to a case in which the red blood-corpuscles were reduced to 1,500,000.

A distinction should be drawn between (*a*) secondary and other forms of anaemia in patients with cirrhosis, and (*b*) cases of Banti's disease or chronic splenic anaemia in which a terminal cirrhosis develops.

Secondary anaemia may be modified by cyanosis which leads to an increase in the number of red cells, hence if cyanosis is due to ascites the blood count may be lowered by tapping; on the other hand, rapid ascitic exudation may by concentration increase the blood count; thus tapping, by favouring the recurrence of ascites, has been observed to be followed by a higher blood count. Hence a patient who is manifestly pale and anaemic may have a relatively high count. Leucocytosis does not occur except from some complication, such as haemorrhage or inflammation. The mean of 22 cases was 6623 (Hanot and Meunier[8]). In Calcutta Rogers[9] finds that leuco-

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[1] Gilbert et Garnier, *Compt. rend. Soc. Biol.*, 1899, li, 59; Gilbert et Weil, *ibid*, 1899, li, 511; De Brynine, *Thèse de Paris*, 1900.

[2] Herrick, F. C. *Journ. Exper. Med.*, N.Y., 1907, ix, 93.

[3] Bouchard. *Rev. de méd.*, 1902, xxii, 837.

[4] Hitschmann. *Centralbl. f. inn. Med.*, 1904, xxv, 42.

[5] Emerson. *Clinical Diagnosis*, p. 586, 1906.

[6] Cabot. *Examination of the Blood*, p. 248, 1897.

[7] v. Limbeck. *Pathology of the Blood*, p. 318. Transl. New Sydenham Soc., 1901.

[8] Hanot et Meunier. *Compt. rend. Soc. Biol.*, Paris, 1895, xlvii, 49.

[9] Rogers. *Lancet*, Lond., 1912, ii, 355.

The venous blood pressure is low in cirrhosis both with and without ascites, and becomes lower after paracentesis (Villaret and Saint-Girons).

Villaret, M. et Saint-Girons, F. *Paris méd.*, 1924, xiv, 471

The [illegible]-content in [illegible] the [illegible]

Among 154 cases of Cirrhosis 74 or 48 per cent., gave a positive Wassermann reaction (Latulle)

The basal metabolism was found to be within the normal limits by AUB and Means who who conclude that the liver is either not an important regulator of the metabolic rate or even when severely damaged is adequate for this purpose.

* Boissonnade and [illegible], Amer. Journ. Med. Sc., [illegible] 304

Latulle Presse méd., Paris, 1918, 477

AUB and Means. Arch. Int. Med., Chicag
1921, xxvii, 173.

cytosis is common and that a high degree is a bad prognostic. The blood serum, as shewn by Garrod's test, may appear to contain uric acid. In advanced cases the alkalinity may be diminished from acidosis. Fiessinger[1] has found that the blood may contain anti-bodies which destroy hepatic cells. Their presence is not constant, but coincides with periods when the cirrhotic process (and the destruction of liver cells which initiates the formation of anti-bodies) is progressing. Welch[2] has noticed marked auto-agglutination of the red blood-corpuscles in some cases.

Respiration.—When there is abdominal distension from ascites or flatulence, the upward displacement of the diaphragm causes shallow and more rapid respirations. Apart from this there is nothing noticeable about respiration, as a rule. The respiration rate may be slowed from a toxaemic state of the nerve centres.

Temperature.—As a general rule, the temperature is not raised; fever is much less frequent in multilobular cirrhosis than in hypertrophic biliary cirrhosis, and should at once suggest the presence of some complication, such as tuberculosis, pleural effusion especially on the right side, malaria, infective endocarditis, or other infections. In 44 cases tabulated by Carrington[3] the temperature was irregular in 18.

Fever is usually associated with hepatic enlargement and sometimes with diarrhoea. It may, in fact, depend on gastro-enteritis, and may then simulate enteric fever or hepatic suppuration (*vide* case on p. 220). A case of cirrhosis with a tongue-shaped lobe and fever has been operated upon for cholecystitis (Henderson[4]).

Apart from manifest secondary infections a febrile temperature is more often seen early in the disease, when its progress is somewhat rapid. Fever is seen in the acute forms of multilobular cirrhosis, and therefore makes the prognosis worse. It may be difficult to decide whether fever in any given case is due to some undetected complication, such as tuberculosis, or to acute progressive changes in the liver.

In a man aged forty-one, whose chart is attached (Fig. 37), admitted for haematemesis, the temperature was almost constantly raised, and in a few weeks ascites developed. Mr. G. R. Turner performed the operation of fixing the omentum between the diaphragm and the liver, and the man greatly improved, and the temperature fell.[5] He died from cirrhosis in a uraemic state about two and a half years later.

Liver.—Clinically the liver is more often enlarged than diminished in size. Foxwell[6] considered that "in the large majority of cases of hepatic cirrhosis at all stages of the disease the liver is felt below the ribs." It has often been assumed that enlargement of a cirrhotic liver depends largely on fat, and that this is more likely to occur in beer drinkers than in spirit consumers. But Foxwell's and my own statistics do not bear out the suggestion that cirrhotic livers of beer drinkers are usually larger than those of spirit drinkers. Further, it is far from universally true that large cirrhotic livers are fatty (*vide* p. 196).

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[1] Fiessinger. *Journ. de physiol. et path. gén.*, Paris, 1908, x, 671.
[2] Welch. *Brit. Med. Journ.*, 1902, ii, 1112.
[3] Carrington. *Guy's Hosp. Rep.*, 1883-4, xlii, 337.
[4] Henderson. *Practitioner*, Lond., 1907, lxxix, 686.
[5] *Vide* Rolleston and Turner. *Lancet*, 1899, ii, 1660.
[6] Foxwell, A. *The Enlarged Cirrhotic Liver*, p. 20, 1896.

In a decided minority of cases, chiefly, I believe, in persons whose history shews no alcoholic excess, the liver is considerably smaller than natural. From ascites, flatulent distension, or from obesity it may be impossible to make out the enlargement. Tympanites often precedes and masks the onset of ascites and prevents an accurate estimation of the size of the liver. Before ascites has appeared the liver may usually be felt, sometimes several finger-breadths, beyond the costal margin in the right nipple line, its surface being firm, slightly irregular, and often tender. In other instances its rough and hard margin can be reached by pushing the fingers under the costal margin, while in other cases, although conditions are favourable, the liver cannot be felt on deep inspiration, and percussion may shew that there is definite diminution in the liver dulness. It should be remembered that percussion is subject to the fallacy that a resonant note may be obtained over the edge of the liver from distended bowel immediately behind. In rare cases the colon may pass in front of the liver and lead to great diminution in the apparent size of the organ. Palpation is therefore more reliable than percussion.

It has been widely assumed that the liver is enlarged in the early

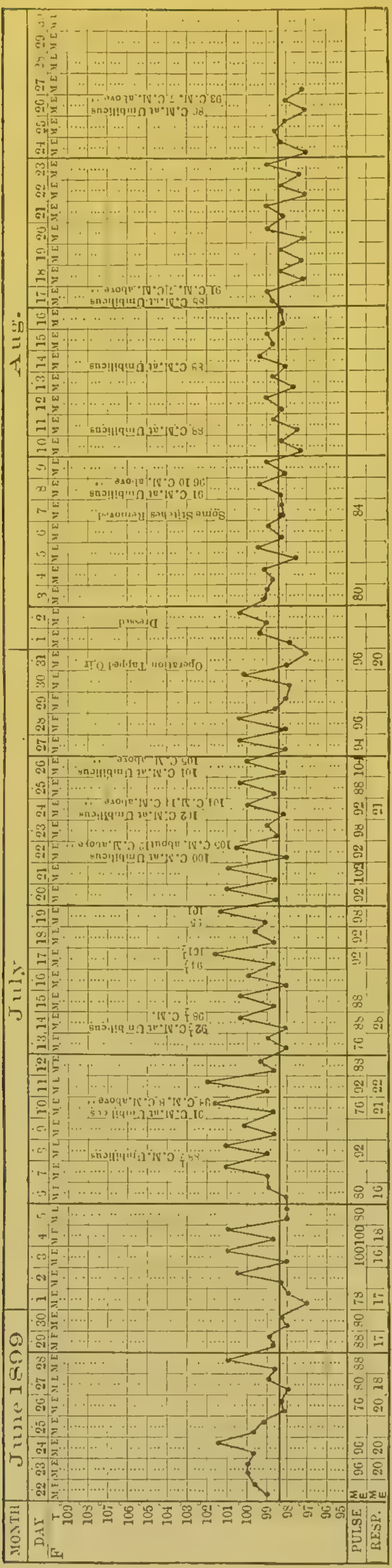

Fig. 37.—Temperature chart from a case of cirrhosis with fever. (*Vide* p. 237.)

stages of the disease and that subsequently it becomes smaller from shrinking and contraction of the fibrous tissue inside it. This sequence of events is sometimes noted (Taylor [1]); thus a considerable time before death the organ has been found to be large, and at the necropsy some months later it has receded behind the costal arch. This may be due to atrophy of areas of compensatory hyperplasia of the lower cells, and not solely to cicatricial contraction. Alterations in the size of the organ may be due to other factors; thus, in the early stage, the enlargement may vary within a comparatively short space of time, thus shewing that the increase in size is due to engorgement. Thus, in alcoholic subjects a drinking bout may lead to rapid increase in size of a cirrhotic liver, the organ feeling firm and being tender, while low diet, total abstinence, and judicious purgation are followed by a return to its former size. Dilatation of the heart due to alcoholic excess, or cardiac failure induced in other ways, may give rise to considerable increase in the size of a cirrhotic liver.

It has been noticed by many observers that the cirrhotic livers of people dying from accident and other causes unconnected with the disease are larger than those of patients dying from the disease.[2] This might be thought to support the view that the liver is enlarged in the early stage from proliferation of the interstitial connective tissue and that later it becomes smaller from cicatricial contraction. But it is more probable that these cases of cirrhosis were latent and that the large size of the livers depended on compensatory hyperplasia of the liver cells, than that the cases were in an early stage of progressive cirrhosis. The surface of the liver may be fairly smooth, especially when it is considerably enlarged, but in other cases its surface is irregular, and sometimes the knobby feel of the "hobnails" is perceptible. These hobnails are not depressed in the centre or umbilicated, but the depressed area of liver substance between two adjacent elevations may imitate the umbilications of multiple carcinomatous nodules.

Hepatic Pain.—Some discomfort and a sense of uneasiness in the region of the liver are very common. Pain is very seldom prominent, but it is present from time to time in a considerable number of the cases, and may be due to transient attacks of perihepatitis, as shewn by the frequency of adhesions between the liver and the diaphragm. Pain of a somewhat severe character over the liver may be due to right-sided pleurisy. Hepatic tenderness is by no means constant; it is often present over large cirrhotic livers after bouts of alcoholic excess, and may be an expression of passive engorgement from failing heart.

Venous Bruits.—In rare instances a venous hum accompanied by a thrill may be heard over the epigastrium; the hum is louder on inspiration. The murmur and thrill may diminish or disappear spontaneously or as the result of tapping, and reappear when the ascites re-collects. There may or may not be enlarged veins visible in the epigastrium. The murmur may be best heard near the umbilicus, and is then thought to arise in a large vein in the falciform ligament. When the hum is loudest over the lower end of the sternum, above the lower margin of the liver, it is probably produced in a dilated coronary vein or in a dilated communication between the internal mammary vein and branches joining the portal vein (*vide* p. 211). The thin-walled and dilated veins may readily be kinked as the result of adhesions or temporarily distorted by the descent of the diaphragm or even by the pressure of a stethoscope. Martini[1] described a soft murmur over the hepatic region, especially in the right axilla, which he regarded to be due to constriction of the inferior vena cava where it passes behind the liver.

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[1] Taylor, F. *Guy's Hosp. Rep.*, 1888, xlv, 310.

[2] Fagge, C. Hilton, *ibid.*, 1875, Ser. iii, xx, 155; Price, J. A. P., *ibid.*, 1883-84, Ser. iii, xxvii, 295.

In a man aged forty-three who was under my care in St. George's Hospital there was a continuous bruit in the epigastrium resembling the *bruit de diable* in the neck. At the necropsy the liver was markedly cirrhotic and the falciform ligament contained a thin-walled vein as large as the little finger (*vide* Fig. 35).

It has also been thought to be due to pressure on the portal vein by enlarged glands, or even to narrowing of the inferior vena cava where it lies in contact with the posterior border of the liver (v. Gambarati[2]).

Spleen.—Enlargement of the spleen is an important feature in portal cirrhosis. It is almost constantly present in fatal cases of progressive cirrhosis, though not so markedly as in hypertrophic biliary cirrhosis. The amount of splenic enlargement has been regarded as an index of the severity of the cirrhosis, and enlargement in cases in which cirrhosis appears to be latent or compensated for should always be an indication for treatment and a cautious prognosis. Considerable enlargement may precede haematemesis, and may therefore be regarded as a danger-signal and call for free purgation. After haematemesis, melaena, or bleeding from piles the spleen may diminish in size. Gilbert and Lereboullet[3] mention cases in which the spleen diminished to one-half or even one-quarter of its previous size after copious haematemesis. In some cases of portal cirrhosis enlargement of the spleen is easily made out, and the organ can be felt to be firm and resistant. In other instances the splenic dulness is increased, though the organ cannot be felt, and in a number of cases the enlargement of the spleen is obscured by tympanites or ascites. Slight pain or discomfort is sometimes experienced in the splenic region, and, apart from the referred pain of flatulence, may be due to inflammation of the capsule, to tension and engorgement of the

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[1] Martini. *Riforma med.*, Napoli, 1894, x, 663.
[2] Gambarati, v. *Ibid.*, 1903, xix, 153.
[3] Gilbert et Lereboullet. *Compt. rend. Soc. Biol.*, Paris, 1904, lvi, 370.

and this opinion also is supported by the occasional occurrence of oedema of other parts of the body, such as the face or arms.

spleen, or to dragging on adhesions between the spleen and neighbouring parts.

Venous Bruits.—As in splenic enlargement from other causes, such as malaria and splenic anaemia, a venous bruit, like the well-known *bruit de diable* in the neck of chlorotic patients, is occasionally heard over the spleen in cirrhosis; according to Catti[1] in one-fifth or one-sixth of all cases of cirrhosis. It has also been compared to the uterine souffle. It may be continuous and increased during inspiration, or may only be heard during inspiration; there may be a palpable thrill. Its position varies, but is generally in the anterior axillary line. It may be due to temporary or prolonged kinking of the dilated splenic vein or of its branches in the hilum of the spleen. Thayer[2] has given a full account of venous murmurs in cirrhosis.

Oedema of the feet usually occurs after, but it may be quite independent of, the onset of ascites; it may even come on in the latest stages, when ascites has passed away and the patient is in a toxaemic state. On the other hand, oedema may begin in the feet before the appearance of ascites. The amount of oedema is usually small; sometimes it is very soft, probably from laxity of the skin and underlying tissues from wasting of the subcutaneous fat and of the muscles. Pre-ascitic oedema of the feet is generally considered to be rather unusual; Silvestri,[3] however, estimates that it occurs in 75 per cent of the cases.

Oedema of the feet is often referred to the ascites mechanically impeding the return of blood through the inferior vena cava. But this explanation will not fit all cases; for example, those in which oedema comes on before the advent of ascites, or when there is so little peritoneal effusion that there can be no increase in the intra-abdominal pressure. In such cases the toxic origin of oedema may be invoked.

It has been thought that the pressure of a firm cirrhotic liver on the inferior vena cava just before it passes through the diaphragm may be sufficient to cause oedema of the feet. This is conceivable, but if it is a factor of any importance, much more oedema of the legs than actually occurs would be expected in cases of malignant disease involving the liver, while a dilated venous plexus, like that seen in mechanical obstruction of the inferior vena cava, should also be present under the skin of the abdominal wall to provide for the return of blood from the lower limbs. Silvestri indeed believes that pre-ascitic oedema of the feet is due to increased pressure in the inferior vena cava brought about by this collateral circulation. As a matter of fact, dilatation of the epigastric and mammary veins may occur when there is ascites, but hardly ever when oedema of the feet is present in the absence of ascites.

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[1] Catti. *Ztschr. f. klin. Med.*, 1907, lxi, 269.
[2] Thayer. *Am. Journ. Med. Sc.*, Phila., 1911, cxli, 313.
[3] Silvestri, T. *Riforma med.*, 1905, xxi, 1101.

In some cases of cirrhosis oedema of the feet may be the outcome of complications, such as the backward pressure of cardiac failure, alcoholism, or valvular disease. Again, it may be due to alcoholic neuritis; ~~in the latter contingency there will be~~ muscular tenderness and absence of the Achillis- and knee-jerks. It is probable that a slight degree of peripheral neuritis is more frequent in the course of cirrhosis than is generally recognised, and that it is specially likely to accompany an aggravation of the liver symptoms with abdominal pain, tympanites, and dyspepsia. When oedema of the feet is associated with considerable ascites, it may be brought about by the pressure of the fluid on the inferior vena cava and the lymphatic vessels going to the thoracic duct.

General oedema is very rare, but the dropsy of the legs and thighs may spread to the abdomen and appear as a lumbar cushion on the back.

McCall Anderson[1] recorded a case of recurrent attacks of universal dropsy in a boy aged eighteen who had no renal disease and whose liver (4 pounds) shewed changes compatible with the view that there was subacute cirrhosis.

ASCITES.—*Derivation.*—ἀσκός, the belly, ὁ ἀσκίτης ὕδρωψ, the abdominal dropsy.

The words "ascites" and "tympanites" have a masculine adjectival termination -ιτης, like the feminine adjectival termination -ιτις in neur*itis* and peritoni*tis*, which agrees with the feminine word νόσος, disease, understood. In the case of *-itis* an entirely secondary meaning, viz. "inflammation," has been evolved.[2]

Definition.—Free fluid in the peritoneal cavity.

Incidence.—Ascites, the association of which with hepatic disease was recognised by Erasistratus (304 B.C.), occurs in 50 per cent of all patients dying with cirrhotic livers. Since it is a late event in the course of the disease it naturally occurs far more frequently in cases fatal from the effects of cirrhosis than in patients who, having cirrhotic livers, die from independent disease, such as pneumonia, tuberculosis, or erysipelas. Ascites occurs in the vast majority of the cases of cirrhosis which run their full course, but a certain number of cases who die, so to speak, prematurely from the direct effects of cirrhosis, such as haematemesis or melaena, do not present ascites.

In Price's[3] 142 cases ascites occurred in 72. In 121 cases of cirrhosis taken from the post-mortem records of the Manchester Royal Infirmary, Kelynack[4] found ascites in 56 per cent. In 166 cases of cirrhosis examined after death at St. George's Hospital ascites was or had been present in 84, or 50·6 per cent. In 80 of these cases death was directly due to cirrhosis, and in 68 of them, or 85 per cent, ascites was present. In the 86 cases fatal from other factors, 16, or 18·6 per cent, had ascites.

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[1] McCall Anderson. *Contributions to Clinical Medicine*, p. 345, 1898, Edin.

[2] Compare Buzzard, T. *Trans. Path. Soc.*, 1889, xl, 347.

[3] Price, J. A. P. *Guy's Hosp. Rep.*, 1883–4, 3. s., xxvii, 295.

[4] Kelynack, T. N. *Birmingham Med. Rev.*, 1897, xli, 86.

Relation between the Weight of the Liver and the Incidence of Ascites.—Ascites is much more often found in association with a comparatively small than with a large cirrhotic liver. This is due to two factors: (*a*) because in an early stage the liver may be larger than it is later in the disease, and (*b*) because when cirrhosis becomes latent from compensatory hyperplasia of the liver cells the organ is considerably increased in size.

Causes of Ascites.—The factors which give rise to the peritoneal effusion in cirrhosis require some discussion. It will be most convenient to refer to them in order and then briefly to summarise their claims.

(I) Mechanical factors: (*a*) Obstruction to the passage of portal blood through the liver. (*b*) Thrombosis of the radicles of the portal vein in the liver. (*c*) Thrombosis of the trunk of the portal vein.

(II) Toxaemia.

(III) Concomitant inflammation of the peritoneum and perihepatitis.

(IV) Associated cardiac failure.

(I) (*a*) *Obstruction to the passage of portal blood through the cirrhotic liver* is undoubtedly present, and appears from Herrick's[1] investigations to depend on the direct communication of arterial pressure to the portal vessels through dilated capillaries, and to the larger volume-flow of the hepatic artery in proportion to the portal flow, as compared to that in the normal liver. It leads to chronic engorgement of the portal vein area, as shewn by the development of a collateral circulation, dilatation, and thickening of the intima of the portal vein, and to some extent clinically also by haematemesis and melaena. The predominance of endothelial cells in the ascitic fluid of uncomplicated cirrhosis is in favour of the view that the ascites is mechanical (Gilbert and Villaret,[2] Ross[3]). But that increased pressure in the portal vein alone does not necessarily produce ascites is suggested by the absence of ascites in cirrhosis at a time when the venous pressure in the portal vein appears to be high, as shewn by haematemesis and melaena. Further, the extreme rapidity with which the effusion may be poured out—a pint a day in some instances—is hardly compatible with the view that it is solely due to increased venous pressure. If the peritoneal effusion was due to the fibrosis in the liver, it would come on gradually, *pari passu* with the morbid changes in the liver, whereas ascites often develops suddenly and rapidly.

Since the collateral circulation is a compensatory means of carrying on the circulation, great importance has been attached to it as preventing ascites. But ascites is often present in spite of considerable collateral circulation, and conversely both ascites and evidence of any portal anastomosis may be absent (Hanot[4]).

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[1] Herrick, F. C. *Journ. Exper. Med.*, N.Y., 1907, ix, 93.
[2] Gilbert et Villaret. *Compt. rend. Soc. Biol.*, Paris, 1906, lix, 820. / l.c
[3] Ross. *Trans. Path. Soc.*, Lond., 1906, lvii, 435.
[4] Hanot. *Arch. gén. de méd.*, 1886, clxxvii, 603.

In Lévi's[1] case of cirrhosis with terminal delirium there were no ascites and no collateral circulation. It is remarkable that there was peritoneal tuberculosis in addition, which in itself is a sufficient cause of ascites.

For these reasons increased venous pressure due to hepatic obstruction, though very frequently associated with ascites, does not entirely account for the peritoneal effusion.

(*b*) It has been suggested that the additional factor required to bring about ascites is thrombosis or obliterating endophlebitis of the terminal branches of the portal vein, either in the liver or where the portal tributaries anastomose with the adjacent systemic veins. There is, however, at present little proof of this.

(*c*) Thrombosis of the portal vein may give rise to ascites, but it is so rare in cirrhosis that it has no claim to be considered among the important causes of ascites.

In 334 cases of cirrhosis examined after death portal thrombosis was present in 10, or 3·3 per cent (Langdon Brown[2]); and in Lissauer's[3] 711 cases in 6, or 0·8 per cent.

(II) *The Toxic Nature of Ascites.*—As a result of hepatic inadequacy poisons absorbed from the alimentary canal or possibly produced elsewhere are no longer stopped and destroyed by the liver, but pass into the general circulation, and a condition of hepatic toxaemia, analogous to that of renal toxaemia, results; the patients become drowsy and may have numerous haemorrhages in various parts of the body. The poisons may either damage the vessels so as to allow increased transudation, or exert a lymphagogue action. It is significant that oedema of the feet may occur before the onset of ascites, inasmuch as this shews that the oedema of the feet is not necessarily a mechanical effect due to the pressure of ascitic fluid on the inferior vena cava. The absence of ascites in biliary cirrhosis, in which there is some reason to believe that the blood is toxic, viz. the considerable splenic enlargement, is perhaps due to the liver cells remaining in a better state of nutrition than in portal cirrhosis, and possibly to the poisons, if present, not being of the same nature as in ordinary cirrhosis.

(III) *Ascites due to Concomitant Inflammation of the Peritoneum.*—In many cases of cirrhosis there is in addition chronic peritonitis, which accounts for the ascites, and especially for ascites which continues for a considerable time or recurs again and again after tapping. Bright, indeed, considered the ascites of cirrhosis to be the result of portal obstruction and of an extension of chronic inflammation from the liver along the portal vein and its branches to the peritoneum. This in a modified form is the opinion held by many authorities, and there is no doubt that ascites is very often satisfactorily explained on this hypothesis. But chronic peritonitis does not account for the onset of ascites in all

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[1] Lévi. *Arch. gén. de méd.*, 1886, clxxvii, 221.
[2] Brown, Langdon. *St. Barth. Hosp. Rep.*, 1901, xxxvii, 62.
[3] Lissauer. *Virchows Arch.*, 1908, cxcii, 278.

Syphilitic peritonitis has been described by
Letulle, and its importance as a cause
of ascites in portal cirrhosis has been
emphasized by Courtois-Suffit and Giroux

It has indeed been thought
that ascites in cirrhosis is
always due to tuberculosis (Roque and Cord

Letulle Presse méd., Par., 1918,
Courtois-Suffit et Giroux, Bull. et mém. Soc. méd des hôp
de Paris, 1919, 3^{e} sér., XLIII, 35

Roque et Cordier. *Rev. de méd.*, Par., 1912, XXXII
76.

cases of ordinary cirrhosis, for it is often absent when there is marked ascites. In some cases ascites passes away when the patient is in a semi-comatose condition; now, since chronic peritonitis would give rise to persistent ascites, such cases may be expected to be free from chronic peritoneal inflammation.

The possibility that ascites is due to a low form of microbic infection will require consideration when more is known about the bacteriology of hepatic cirrhosis. This suggestion is attractive from the fact that bacterial infections may occur in other parts of the body in the later stages of cirrhosis. From analogy it appears probable that the peritoneum would suffer similarly, since infection from the intestinal tract, which is often in a condition of chronic catarrh, might easily occur. Again, ascites may supervene after infective processes have occurred elsewhere in the body, thus suggesting that the ascites is in some way secondary.

Although as seen in laparotomies on cases with ascites the peritoneum is highly injected, there is no inflammation or acute peritonitis. So if there be a microbic origin for ascites, the micro-organisms are of a low degree of virulence. Tuberculosis of the pleura and peritoneum may give rise to almost pure serous effusions; and in these cases the fluid withdrawn may appear to be almost, if not quite, free from bacilli until inoculated into guinea-pigs. Possibly the same may hold for ascites of cirrhosis, viz. that it is due to a micro-organism which does not appear in the fluid withdrawn by paracentesis.

Ascites due to Concomitant Perihepatitis.—Some thickening of the capsule of the liver is frequent in cirrhosis, and in some instances satisfactorily explains ascites. But it is not a constant cause, for ascites may occur when there is little or no perihepatitis, and it may be absent when there is as much perihepatitis as there is in cases in which the peritoneal effusion is thus explained. Parkes Weber[1] suggested that effusion resulting from localised areas of inflammation may possess sufficiently irritating properties to lead to a copious exudation of normal or nearly normal fluid from the non-inflamed surfaces. On this view quite local perihepatitis might account for ascites. Against this view is the occurrence of foci of perihepatitis in the absence of ascites, both in cirrhosis and in other conditions. Weber[2] has also suggested that transient ascites in cirrhosis may be due to local peritonitis which is temporary.

(IV) *Ascites from Cardiac Failure.*—In cases of latent cirrhosis dilatation of the heart from valvular disease, from affections of the myocardium, whether chronic or acute, or from obstructive lung disease may develop and give rise to ascites. The cirrhosis and ascites are therefore associated, but the ascites is not necessarily dependent on the cirrhosis. On the other hand, backward pressure very probably favours the onset of ascites in patients with cirrhosis; in other words, an amount of backward pressure which in an ordinary case would not produce ascites may be the cause of ascites in a patient with progressive cirrhosis. Heart disease may therefore induce ascites in cirrhosis in two ways: (i) Merely by backward pressure, as in some cases of tricuspid regurgitation; (ii) by bringing about conditions, such as venous engorgement, the stagnation of toxins, or damage to the endothelial cells of the peritoneum, which precipitate ascites due to cirrhosis.[1]

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[1] Weber, F. P. *Brain*, 1902, xxv, 150.

[2] *Idem.* *Edin. Med. Journ.*, 1903, N.S., xiii, p. 322.

In conclusion, ascites may be merely associated with cirrhosis and directly due to chronic or possibly to subacute peritonitis and perihepatitis, or to cardiac failure; in rare cases it may be due to a direct complication of cirrhosis, *i.e.* thrombosis of the portal vein, or, on the other hand, it may be due to cirrhosis alone. In the latter case it may be explained as being partly due to portal obstruction which provides the vascular conditions favourable to transudation, and partly to the presence of toxins in the blood which damage the vessel walls or act as lymphagogues and so induce an exaggerated flow of fluid into the peritoneal cavity. It is probable that this form of ascites may be brought about prematurely by cardiac failure.

The **onset of ascites** may follow so soon after some event, such as a blow, fall, or exposure to cold, that there would appear to be a definite connexion between them. It is possible that by a blow the resistance of the peritoneum may be so reduced that the real responsible cause of ascites is enabled to produce its effect sooner than it otherwise would.

In a boy aged twelve years a radical cure for hernia was rapidly followed by ascites; this was tapped eight times; at the necropsy the liver (27 ounces) was extremely cirrhotic; there was no chronic peritonitis, but there were some adhesions from a past attack of acute peritonitis.

As the result of a chill, vasomotor paralysis of the splanchnic vessels results, and might thus precipitate ascites, so to speak. These cases have been spoken of as acute ascites.[2] It must be remembered that what appears to be acute ascites may prove to be acute peritonitis. Ascites may come on after various infections, such as suppuration, erysipelas, influenza, or typhoid.

Though it may be sudden, the onset of ascites is usually gradual, and is often masked by flatulent distension of the abdomen. It thus contrasts with the sudden and rapid development of ascites in thrombosis of the trunk of the portal vein. Ascites occurs late in the course of uncomplicated cirrhosis, that is, in cases in which there is no concomitant chronic peritonitis. The onset of ascites in such cases is a grave event, as the patient seldom survives long enough to require tapping more than two or three times; Hale White says the patients do not live long enough to require more than one tapping, but in my experience this view is too extreme.

Characters of Ascitic Fluid.—The ascitic fluid is clear, greenish or

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[1] Compare Cheadle, W. B. *Some Cirrhoses of the Liver*, p. 103, Lond., 1900.

[2] B. Weil (Acute Ascites), *Thèse de Paris*, 1899, No. 278; Potain, 'Ascites a frigore," *Sem. méd.*, 1888, viii, 9.

In 45 cases of ascites due to uncomplicated cirrhosis, 31 required tapping and on an average this was done twice (Rambottom).

but a high lymphocyte
count may occur in
uncomplicated Cirrhosis (Cabot);
I have seen several examples
of this.

Cabot. Journ. Am. Med. Assoc., Chicago,
1912, LIX, 2295

yellow in tint, and sometimes slightly bile-stained. Its reaction is alkaline and its specific gravity between 1008 and 1015. It contains more albumin than ascitic fluid of renal disease and less than that of cardiac ascites (Dickinson[1]). If there is any added peritonitis, its specific gravity and the proportion of albuminous matter increase and flakes of coagulated fibrin may form on standing. It contains from 0·5 to 4·4 per cent of albuminous material and occasionally a trace of sugar, urea, urobilin. The large amount of chlorides in ascitic fluid is associated with the diminished output in the urine. If the specific gravity is above 1015, contains more albumin, and microscopically shews polymorphonuclear leucocytes, there is some inflammatory change in the peritoneum.

Character of the Cells in the Ascitic Fluid.—In uncomplicated cirrhosis the endothelial cells are present in predominating numbers (Cade,[2] Ross,[3] Gilbert and Villaret[4]) and may constitute 95 per cent of the cells. As the result of several punctures the percentage of endothelial cells may fall owing to infection (Gilbert and Villaret). In cases with an equal percentage of endothelial cells and lymphocytes tuberculous infection ~~should~~ be suspected, A predominance of lymphocytes/ ~~points to~~ tuberculous peritonitis. ~~Grenet and Vitry[5] found endothelial cells and a few lymphocytes. Souques,[6] and Achard and Laubry,[7] in two cases of lactescent ascites in cirrhosis, found lymphocytes chiefly represented.~~ may

Chylous ascites due to an admixture of chyle may occur in cirrhosis.

Wallis and Schölberg[8] collected 13 cases in which cirrhosis was associated with chylous ascites; in Merklen's[9] case chylous ascites followed a fall, and thus suggests laceration of lymphatic vessels, though this was not made out at the necropsy. In Nichols'[10] case the receptaculum chyli was thrombosed.

Resembling true chylous ascites in appearance, but differing from it chemically, are the two following forms: (*a*) the chyliform or fatty, and (*b*) the milky non-fatty ascites.

In chyliform or fatty (adipose) ascites the fat, which is present in larger globules than in the chylous ascites, is probably the outcome of degenerative changes in cells suspended in the ascitic fluid. In cases of fatty ascites in cirrhosis, Souques and Achard and Laubry found both fat and a nucleo-albumin; the latter gave rise to an opalescent solution. Fatty ascites would thus differ from the next form only in the presence of fat, since in both there may be a nucleo-albumin capable of rendering ascitic fluid milky. Fatty ascites has been thought to be due to lipaemia, and in a case of cirrhosis the fibrotic changes in the pancreas were suggested as the underlying factor (Gaultier[1]).

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[1] Dickinson, W. H. Allbutt's *System of Med.*, 1898, v, 668.
[2] Cade. *Arch. de méd. expér. et d'anat. path.*, Paris, 1906, xviii, 769.
[3] Ross, E. A. *Trans. Path. Soc.*, Lond., 1906, lvii, 435.
[4] Gilbert et Villaret. *Compt. rend. Soc. Biol.*, Paris, 1906, lix, 820. l.c.
~~[5] Grenet et Vitry. *Sem. méd.*, 1903, xxiii, 235.~~
[6] Souques. *Bull. Soc. méd. d. hôp. de Paris*, 1902, 3. s., xix, 290.
[7] Achard et Laubry. *Ibid.*, 295.
[8] Wallis and Schölberg. *Quart. Journ. Med.*, Oxford, 1910–11, iv, 170.
[9] Merklen. *Sem. méd.*, 1897, xvii, 181.
[10] Nichols, H. J. *Med. News*, N.Y., 1905, lxxxvii, 925.

In opalescent, milky (non-fatty) ascites, of which Jousset[2] has collected 14 cases in portal cirrhosis, there are a large number of mononuclear leucocytes which probably give rise, as the outcome of degeneration, to some body, such as nucleo-albumin or globulin, which is responsible for the lactescent appearance of the fluid. It has, however, been suggested that lecithin may cause the milky character. No fat can be extracted by ether from the ascitic fluid in this form. In both the fatty and the non-fatty forms of milky ascites the change may appear on the second tapping, in cases in which the fluid withdrawn on the first occasion was clear.

Haemorrhagic ascites is very rare in cirrhosis. It is not due to hepatic insufficiency, like the oozing from the gums and epistaxis. Neither is it usually due to concomitant tuberculosis of the peritoneum. It may be the result of previous tapping; thus, in cases in which the fluid is serous at the first paracentesis and sanguineous at subsequent tappings, it is probable that the trocar wounded some of the vascular adhesions or dilated veins in the peritoneum (Barjon and Henry[3]). In some instances of cirrhosis chronic haemorrhagic peritonitis occurs and possibly may be due to, at any rate is associated with, alcoholism (Fernet[4]). Acute infection, for example with streptococci, as in a case recorded by Bauer,[5] may render haemorrhagic the ascites of chronic peritonitis associated with cirrhosis.

Bacteriology of Ascitic Fluid.—More observations are required as to the bacteria present in the ascites of cirrhosis, and very probably may be obtained by the method of inoscopy. It consists in taking the coagulum of the fluid; or, if there is no spontaneous one, producing one, dissolving it with aseptic gastric juice, and examining for micro-organisms; this method depends on the fact that the clot filters off the micro-organisms from the fluid. By this method Jousset[6] found tubercle bacilli in many cases thought to be cirrhosis, which is quite in accord with the comparative frequency of tuberculous peritonitis in cirrhosis.

M. Abbott[7] found a colon bacillus, of the same type as Adami's minute diplococcus, in the ascitic fluid of three cases of cirrhosis. In one instance, in which there was chronic peritonitis as well as hepatic cirrhosis, this micro-organism was isolated from three tappings during life.

Tension of Ascitic Fluid.—There may be a positive pressure inside the abdomen in ascites, as shewn by the upward displacement of the diaphragm.

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[1] Gaultier. *Compt. rend. Soc. Biol.*, Paris, 1906, lix, 429.

[2] Jousset. Quoted by Souques.

[3] Barjon et Henry. *Lyon méd.*, 1898, lxxxviii, 258.

[4] Fernet. *Bull. Soc. méd. d. hôp. de Paris*, 1900, 3. s., xvii, 781.

[5] Bauer. *Arch. gén. de méd.*, 1905, cxcv, 1351.

[6] Jousset. *Arch. de méd. expér. et d'anat. path.*, 1903, xv, 289.

[7] Abbott, Maude. *Journ. Path. and Bacteriol.*, 1900, vi, 315.

Pitres[1] found that it may vary between 30 and 6 millimetres of mercury, the average positive pressure being about 12 millimetres.

This positive intra-abdominal pressure varies with respiration and is associated with increased pressure in the portal vein and low arterial pressure (Gilbert and Weil[2]).

Physical Signs.—The abdomen is greatly enlarged; its girth may be 50 inches or even more. The enlargement, which is fairly uniform, is more marked antero-posteriorly at first; when it has lasted for some time there is bulging in the flanks. The parietes are stretched and the skin often shews the effects of distension in lineae albicantes, which are especially visible after paracentesis. The abdominal muscles become lax and atrophied and the umbilicus may be everted and project like a tense thin-walled bladder; it may ulcerate, and has been known to burst. In a man under my care who had been tapped several times the thin-walled bulla at the umbilicus became the site of a Littré's hernia. The veins over the umbilicus are sometimes very prominent. The loss of tone in the abdominal parietes favours flatulent distension, especially after the intra-abdominal pressure has been reduced by paracentesis. The costal margins are pushed forwards and the ensiform cartilage may be carried upwards and forwards. The subcutaneous veins of the abdominal walls become prominent and enlarged. There are two sets of veins: (*a*) Those around and above the umbilicus, which form part of the compensatory anastomosis between the veins of the abdominal wall and the parumbilical veins in the falciform ligament. A dilated and varicose condition of the veins around the umbilicus has been called cirsomphalos. (*b*) The veins running up from the middle of the groin (the superficial and deep epigastric veins) towards the middle of the costal arch (superior epigastric and long thoracic veins). Dilatation of these veins points to obstruction to the passage of blood through the inferior vena cava. A large ascitic effusion may, by increasing the intra-abdominal pressure, seriously interfere with the return of blood from the lower extremities, and so lead to opening up of this anastomotic channel. Hence in considerable ascites both these sets of veins are manifestly enlarged; removal of the ascitic fluid is followed by disappearance of the "caval," while the "portal" veins remain (Gilbert and Villaret[3]).

The ascitic fluid presses the diaphragm up and diminishes the capacity of the thorax. The heart may thus be so displaced upwards that the apex beat is in the third interspace. This displacement may be associated with a systolic murmur over the pulmonary artery, due to twisting or kinking of its trunk. When the displacement is corrected by tapping this murmur disappears.

The liver dulness is displaced upwards, and anteriorly may reach to the fourth, third, or even second rib, while posteriorly the resonance over the bases of both lungs is also encroached upon. If there is dulness over the lower lobe of the right lung alone, the question arises whether this is due solely to upward displacement of the liver and collapse of the lung or whether there is, as often occurs (*vide* p. 285), an effusion into the right pleural cavity. When the patient takes a deep breath, the dulness is unaltered if there is an effusion, but will diminish somewhat if due solely to the liver; in pleural effusion the dulness is higher in the axilla, while the dulness due to the liver is more marked behind; when the patient lies on his face dulness due to the liver diminishes or disappears.

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[1] Pitres. *Compt. rend. Soc. Biol.*, Paris, 1899, li, 674. ll.c

[2] Gilbert et Weil. *Ibid.*, 1899, li, 511.

[3] Gilbert et Villaret. *Rev. de méd.*, Paris, 1907, xxvii, 334.

In women, the uterus is pressed forwards by the fluid in Douglas' pouch, and depressed, and may thus become prolapsed.

The fluid collects in the flanks, which are dull on percussion. In order to distinguish ascites from faecal distension of the colon the patient should be turned over, when the note will become resonant; this shifting dulness is proof of free fluid in the peritoneal cavity. It should be pointed out that in a certain proportion of cases of ascites there is resonance in one or both flanks, due to gaseous distension of the colon. The absence of dulness in the flanks, therefore, must not be taken as absolute proof against ascites and in favour of an ovarian cyst. After collecting in the flanks the fluid rises out of the pelvis and gives rise to a dull note on percussion. This dull area gradually increases in extent and approaches the umbilicus. The stomach and intestines are floated upwards against the abdominal parietes in the middle line between the umbilicus and the ensiform cartilage. The area of resonance eventually roughly corresponds with that of the epigastric region. Chronic peritonitis may be combined with cirrhosis, and if well marked may lead to such retraction of the mesentery that the intestines, being more or less tethered to the spine, are unable to reach the anterior abdominal wall; in such cases there may be complete absence of resonance over the front of the abdomen.

In definite ascites a thrill is readily produced by flipping the abdominal wall in one flank with the finger; a distinct impulse is then felt by the other hand laid flat on the opposite side. In order to prevent the impulse from being transmitted through the abdominal walls, an assistant should place his hand or a piece of cardboard on the linea alba in the long axis of the body. When the abdominal wall is laden with fat or swollen from subcutaneous oedema, the precaution is especially necessary. In the presence of old peritoneal adhesions, the ascitic effusion will very probably become encysted. The physical signs may then resemble those of a fixed cyst, and are very difficult to interpret correctly. Encysted ascites is, however, rare in connexion with hepatic cirrhosis.

When there is free fluid in the peritoneal cavity, it is found under certain conditions that if the hand is placed flat over the liver and a sudden sharp flexion of the fingers made, a sensation of displacement of fluid is produced and the finger-tips come in contact with the firm liver. This sign—"dipping for the liver"—cannot always be obtained. It depends on the presence of fluid between the liver and the abdominal wall. Hence adhesions between the liver and the parietes, or the fact

From analysis of 16 cases Cabot found that on an average the fluid reaccumulated at the rate of a pint a day. ^

Cabot. *Am. Journ. Med.* Sc., Phila., 1912, CXLIII, 1.

that the liver is in immediate contact with the abdominal wall, prevent this manifestation of ascites.

Symptoms and Effects of Ascites.—There is a feeling of tightness and discomfort in the tense and stretched abdominal walls. The upward displacement of the diaphragm causes collapse of the lower lobes of the lungs, and so dyspnoea, and even orthopnoea. There is often some bronchitis, and in rare instances haemoptysis may result from the engorged and collapsed lung. When, with increasing ascites, signs of oedema of the lower lobes of the lungs appear, the abdomen should be tapped without delay.

The upward displacement of the heart may be accompanied by irregularity, palpitation, or faintness. The pressure of ascites on the kidneys and their vessels may help to curtail the excretion of urine and possibly to produce albuminuria, but the diminished amount of urine is chiefly due to the low arterial pressure. Oedema of the legs may be accelerated by the pressure of the peritoneal effusion on the inferior vena cava, but it is not solely due to this mechanical cause, inasmuch as oedema of the feet may precede the appearance of ascites, or indeed occur independently. As is pointed out elsewhere, toxaemia plays an important part in the production of oedema of the feet (*vide* p. 241).

If ascites becomes excessive, the umbilicus, which has become everted and thin, may burst; this spontaneous discharge has also been known to take place through the cicatrix of an umbilical hernia (Merklen and Gougelet[1]). Such an event should never be allowed to occur; not only is the extreme distension very harmful, but the rupture of the abdominal wall may introduce infection, and so set up fatal peritonitis. Most of the recorded cases occurred years ago, when paracentesis was postponed as long as possible.^

Differentiation of Ascites from other Abdominal Conditions.—A large, thin-walled *ovarian or parovarian cyst*, if so large as to fill a great part of the abdomen, may cause great difficulties in diagnosis. In the case of an ovarian cyst the history may shew that the swelling definitely began in one situation, that it arose out of the pelvis and then spread upwards. The outline of the cyst may be felt, especially on deep inspiration. In a doubtful case a vaginal examination may reveal a cyst with pelvic attachments. The abdomen is prominent in an antero-posterior direction, while in simple ascites the bulging is also in a lateral direction. The dulness is in or near the middle line of the abdomen and not in the flanks; but, as already pointed out, resonance in the loin may persist in ascites. The maximum abdominal girth is below the umbilicus, while in ascites it is at or above this level. In ascites the umbilicus is in its normal position, viz. one inch nearer to the pubes than to the ensiform cartilage, while with an ovarian cyst this relation may be greatly altered in the opposite direction. When an ovarian cyst is on either side of the middle line, it may displace the umbilicus, so that it is nearer one anterior superior spine than the other. This displacement may only be revealed by careful measurement, and not be manifest on mere inspection. In ovarian cysts the thrill may be absent, and when present is less marked than in ascites. There is little constitutional disturbance in ovarian cysts, the ill effects being almost entirely due to mechanical pressure. When an ovarian cyst is complicated by ascites, the diagnosis is extremely difficult. Rupture of the cyst with extravasation of its contents into the peritoneal cavity may occur spontaneously in thin-walled cystadenomas and parovarian cysts, or from trauma. If the fluid is non-irritating, it is absorbed, with the result that polyuria follows; but if irritating, peritonitis is set up.

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[1] Merklen et Gougelet. *Bull. Soc. méd. d. hôp. de Paris*, 1901, 3. s., xviii, 952.

Solid or Semi-solid Abdominal Tumours simulating Ascites.—Fatty, fibro-fatty, and myxo-lipomatous tumours, though comparatively rare, may, when they grow to a considerable size—and some of them have weighed as much as 40 pounds—very closely imitate ascites. In fact, many of the recorded examples have been tapped fruitlessly, and several of them more than once. Of the fatty tumours, of which Proust and Trèves[1] collected 89 examples, about one-third arise from the fat around the kidneys, the remainder take origin from the retroperitoneal and other tissues. The tumours, whether originally fatty or fibrous, are prone to become oedematous and undergo myxomatous degeneration and so readily fluctuate.

Diffuse colloid carcinoma or endothelioma of the peritoneum is very rare; it may give rise to great enlargement of the abdomen and to dulness on percussion, but fluctuation and thrill are not present unless there is concomitant ascites. In some cases there is no difficulty in feeling definite tumours, and then there is no resemblance to ascites. In Pye-Smith's[2] case of colloid carcinoma involving the peritoneum tapping was performed thirty-six times.

Encysted peritonitis and the localised tuberculous peritonitis in the lower and front portions of the abdomen[3] (Tait's "allantoic cysts") differ in the extent of the peritoneal cavity involved and in the fact that the dulness due to the effused fluid does not alter with changes in the position of the patient.

Hydatid Cysts.—Murchison described an enormous cyst starting from the liver and passing through the foramen of Winslow into the general peritoneal cavity, which it almost filled. In its fully developed or latest stage it resembled ascites, but the history shewed that the abdominal swelling was at first localised to the right side.

Various abdominal cysts, such as pancreatic, omental, chylous, urachal, mesenteric cysts, are seldom, if ever, so large as to resemble ascites. A large pyonephrosis or hydronephrosis is so unilateral that it should not be mistaken for ascites. In very exceptional instances a dilated stomach, a dilated gall-bladder, and a very large hepatic abscess (Powell,[4] Hatch[5]) have been diagnosed as ascites.

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[1] Proust et Trèves. *Rev. de gyn. et de chir. abdom.*, 1908, xii, 93.

[2] Pye-Smith. *Trans. Path. Soc.*, 1893, xliv, 116.

[3] Doran, A. *Med.-Chir. Trans.*, Lond., 1898, lxxxi, 320.

[4] Powell. *Ind. Med. Gaz.*, 1898, xxxiii, 41.

[5] Hatch. *Ibid.*, 1898, xxxiii, 205.

From a Pregnant Uterus with Hydramnios.—The outline of the enlarged uterus may be made out by palpation through the abdominal walls. Other signs of pregnancy, such as mammary enlargement and engorgement, a soft and patulous os uteri, should be looked for.

Obesity.—When the abdominal walls are laden with fat, the detection of a small amount of ascitic fluid is difficult and often impossible. On the other hand, a feeling of semi-fluctuation may be obtained through the pendulous fatty walls and suggest ascites. To control this the hand of an assistant should be placed with the ulnar side downwards on the abdominal wall between the two hands of the observer. Inspection alone should assist in the diagnosis. When there is much fat in the abdominal parietes, the umbilicus is buried, whereas in ascites it tends to be everted.

Diagnosis of Ascites due to Cirrhosis from that due to other Causes.—Under the following heads attention will chiefly be paid to the main points that are in favour of the various conditions which may imitate cirrhosis by inducing ascites.

Acute peritonitis can hardly be confounded with the ascites due to cirrhosis, for the onset is sudden, the constitutional symptoms are prominent, and the abdominal distension is due to paralytic dilatation of the intestines with gas rather than to the quantity of fluid. It should be mentioned, however, that ascites ~~may~~ sometimes comes on rapidly in cirrhosis, especially when complicated by portal thrombosis. Unlikely as it would appear, the sudden onset of ascites in a case of alcoholic cirrhosis with subacute gastritis has simulated intestinal obstruction.

The ascites due to the various forms of *chronic peritonitis* is extremely likely to be mistaken for that of cirrhosis. Chronic peritonitis and universal perihepatitis (*vide* p. 161) frequently cause ascites, usually in association either with adherent pericardium or with arteriosclerosis and granular kidney. In a doubtful case albuminuria is therefore against the existence of cirrhosis as the primary cause. In chronic peritonitis the ascites recurs again and again after tapping; cases are on record in which the abdomen has been tapped fifty or even a hundred times. This is the most reliable criterion in distinguishing chronic peritonitis from cirrhosis. Enlargement of the spleen, which is comparatively frequent in cirrhosis, is not a prominent feature in chronic peritonitis, unless there is some complication, such as lardaceous disease. Haematemesis hardly ever occurs in chronic peritonitis; hence the association of haematemesis and ascites points to cirrhosis. In cases in which haematemesis is associated with frequently recurring ascites chronic peritonitis is probably complicated by some other condition, such as gastric ulcer or cirrhosis.

Tuberculous peritonitis may supervene in the course of cirrhosis, and is then very likely to be regarded as simple ascites. In 584 cases of common cirrhosis peritoneal tuberculosis was present in 53, or 9 per cent (*vide* p. 223). The primary lesion here is cirrhosis, which disposes the peritoneum to tuberculous infection. In these cases the liver is markedly cirrhosed, while the peritonitis is of more recent date. Although in tuberculous peritonitis a slight secondary hepatic fibrosis is sometimes seen, it is microscopic rather than macroscopic.

The mistake that has often occurred is to do laparotomy on a child with cirrhosis and ascites in the belief that it is tuberculous peritonitis. If such a thing occurs, it would be advisable to take the opportunity of uniting the liver and great omentum to the abdominal wall so as to lead to vascular adhesions (*vide* p. 260).

Tuberculous peritonitis in adults, except when secondary to cirrhosis, is most commonly seen in women, and often spreads from the uterine appendages. The following points are in favour of tuberculous peritonitis: evidence of tuberculosis elsewhere; abdominal pain and tenderness, especially in the lower part of the abdomen and about the right iliac fossa; pain on micturition; fever; enlarged glands in the abdomen or groin; a moderate degree of effusion; a hard cord due to the rolled-up omentum; induration, redness of the skin, and evidence of a small abscess around the umbilicus. When drawn off, the fluid is more turbid, of a higher specific gravity (1020 instead of 1010), and richer in albumin than in cirrhosis. A preponderance of lymphocytes points to tuberculosis; the presence of numerous lymphocytes and of some endothelial cells is in favour of the association of cirrhosis and tuberculous peritonitis. Ordinary examination for tubercle bacilli is often negative, but injection into guinea-pigs gives rise to tuberculosis. The method of inoscopy,[1] or bacteriological examination of the clot formed in the fluid, may give positive results when ordinary methods fail (*vide* p. 248).

In ascites due to multiple nodules of malignant disease in the peritoneum a primary source may be manifest in the stomach (symptoms of dyspepsia, pyloric obstruction, absence of HCl in vomit), in the rectum, and in women in the pelvic organs. The umbilicus may be hard and infiltrated with growth, and sometimes small subcutaneous tumours can be felt in the line of the falciform ligament or elsewhere in the abdomen. Small masses of fat, however, may very closely imitate these minute secondary growths. In malignant disease there is more emaciation and pigmentation of the face is more frequent than in cirrhosis. The spleen is not enlarged and there is no history of haematemesis. Sometimes the glands in the groin are enlarged, and occasionally multiple subcutaneous tumours are present.

In *malignant disease of the liver* the presence of deep jaundice together with ascites, the enlargement and irregularity of the liver, the progressive hepatic enlargement and more rapid emaciation, help to differentiate it from ascites due to cirrhosis. Other points in favour of malignant disease are evidence of growth elsewhere, induration of the umbilicus, and absence of splenic enlargement. In cases with ascites it is often impossible to distinguish between cirrhosis and malignant disease of the liver until the abdomen has been tapped. A large nodular liver points to new-growth.

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[1] Jousset. *Arch. de méd. expér. et d'anat. path.*, 1903, xv, 289.

, which is more
marked in the
ascitic fluid than
in the blood serum
(ESMAIN and Parvu),

ESMEIN ~~and~~ et PARVU. *Compt. rend. Soc. biol.*,
Par., 1909, LXVI, 159

Syphilitic disease of the liver may cause ascites. ~~The presence of~~ syphilitic lesions elsewhere and a positive Wassermann reaction, call for antisyphilitic treatment, which may clear up the diagnosis. When enlarged from gummatous or lardaceous change, or from both combined, the liver is firm and may be irregular, and may be mistaken for malignant disease.

Lardaceous disease of the liver does not often give rise to ascites in adults. The following case of lardaceous liver closely imitated cirrhosis :

A woman aged forty-seven under my care in St. George's Hospital, June-July 1900, had the facial aspect of cirrhosis; there were signs of chronic pulmonary tuberculosis at both apices, oedema of the feet which preceded by two weeks the onset of ascites, and albuminuria. There was a history of haematemesis which in the light of the necropsy was probably haemoptysis. The ascites required tapping twice ; the liver and spleen could not be felt. The patient was thought to have cirrhosis. The patient died semi-comatose. The necropsy shewed lardaceous liver (53 ounces) and kidneys, due to chronic pulmonary tuberculosis, with three or four vomicae in both upper lobes. There were slight thickening of the capsule of the liver and opacity of the peritoneum, but not enough chronic peritonitis to account for the ascites. There was no cirrhosis of the liver.

The history of past suppuration or of syphilis, evidence pointing to lardaceous disease of the kidneys (albuminuria) or of the intestines (diarrhoea), are important points in making a diagnosis.

Thrombosis of the portal vein is rare, but when it does occur is probably associated most often with portal cirrhosis of the liver (*vide* p. 56). When the trunk of the portal vein is affected, ascites, if not already present, rapidly develops. The spleen enlarges from obstruction of the splenic vein, and haematemesis may occur. The severity of the symptoms and their onset in a person whose past history shews neither cause for nor evidence of cirrhosis would be in favour of primary thrombosis of the portal vein. But it will always be difficult to be sure that the case is not one of latent cirrhosis. The difficulty of a correct diagnosis in some cases of portal thrombosis supervening in the course of cirrhosis is illustrated in the following case :

A man aged fifty-six, in a state of great weakness and considerable emaciation, was admitted under me at St. George's Hospital complaining of "dropsy, stoppage of the bowels, and difficulty in passing water." He had noticed oedema of the feet five weeks before ; this was followed by constipation and difficulty in passing water. He had been quite a moderate consumer of beer, and denied syphilis. He was found to have marked ascites ; nothing else abnormal could be felt in the abdomen, and he was thought to be probably the subject of malignant abdominal disease. He died thirty-six hours after admission. At the necropsy there were chronic peritonitis, marked portal cirrhosis, and thrombosis of the portal vein ; the liver weighed 4 pounds and the spleen 4 ounces.

Ascites from ovarian papilloma may recur very frequently, and thus differs from the ascites of uncomplicated cirrhosis. The diagnosis can be clinched by vaginal or rectal examination and the detection of irregular masses of growth choking up the pelvis, or by the recognition of pieces of the characteristic villous growth in fluid withdrawn from the abdomen.

Pye-Smith[1] recorded a remarkable case of papillomatous tumours of both ovaries which gave rise to ascites in a woman aged thirty-five, lasting nine years and necessitating 299 tappings. At the necropsy there were numerous implantation growths over the parietal peritoneum.

Ascites due to backward pressure of tricuspid regurgitation, etc., should be recognised by examination of the heart and the recognition of mitral or obstructive lung disease—emphysema, chronic interstitial pneumonia, or pneumonokoniosis. In some cases of cirrhosis failure of the heart, such as may occur from alcoholism, may induce ascites, so that cirrhosis and ascites are associated, though not related as cause and effect. In such cases it is difficult to decide their relation until the alcoholic dilatation of the heart has been successfully treated.

In *renal disease* ascites is part of the general dropsy which typically affects the face. The character of the urine and the cardio-vascular changes proper to renal disease should prevent any mistake in diagnosis. As pointed out elsewhere (p. 224), renal disease may coincide with portal cirrhosis.

In *splenic anaemia* of adults ascites very occasionally appears before the terminal cirrhosis of the liver, which is known as Banti's disease, has developed, as shewn by post-mortem examination of cases of uncomplicated splenic anaemia. Inasmuch as periodic haematemesis also occurs in that disease, the resemblance to cirrhosis when ascites develops might be thought to be very close. Clinically the anaemia and splenic enlargement are so much greater in splenic anaemia that ordinary cirrhosis is only likely to be diagnosed by those who are not familiar with the fact that ascites and periodic attacks of haematemesis may occur in splenic anaemia.

The **prognosis** of ascites due to uncomplicated cirrhosis is very bad; as a rule, death follows within a short time of its onset—usually within three to four months, and sometimes much sooner.

A distinction has rightly been drawn, more especially by Hale White[2] and Campbell Thomson,[3] between ascites due to cirrhosis and ascites associated with cirrhosis, but due to some other cause, such as chronic peritonitis. In ascites associated with chronic peritonitis tapping may be required frequently, and occasionally recovery may occur; whereas in ascites due to cirrhosis tapping is seldom required more than two or three times, and is followed by death sooner than in the first category.

1| In 31 cases of uncomplicated cirrhosis the average number of days of survival after the appearance of ascites was ~~[illegible]~~; ~~in 31 of these cases paracentesis was necessary~~ 188, and the average duration of life after the first tapping was 46

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[1] Pye-Smith. *Trans. Path. Soc.*, 1893, xliv, 111.
[2] Hale White. *Guy's Hosp. Rep.*, 1893, xlix, 1.
[3] Thomson, Campbell. *Med.-Chir. Trans.*, Lond., 1901, lxxxiv, 251.

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days. Whereas in 1[illegible] cases of cirrhosis, complicated with chronic peritonitis, the average number of days for the same interval came to ~~300~~ and 288 (Ramsbottom[1]).

It is, however, possible that in some cases of cirrhosis with chronic peritonitis and ascites, the ascites causes the chronic peritonitis, and not vice versa. A persistent ascites might in virtue of contained toxins or bacteria set up some chronic peritonitis. If this is so, the prognosis of ascites in cirrhosis is not so gloomy as is usually supposed. Since oedema of the feet in the course of cirrhosis is a sign of general toxaemia, the prognosis in a case of cirrhosis with ascites and oedema of the feet is worse than in cirrhosis with ascites alone, inasmuch as the latter may be a case of cirrhosis associated with ascites due to some other factor, such as chronic peritonitis. The presence or absence of oedema of the feet may thus be useful in determining whether, in any case of ascites due to cirrhosis, the termination is likely to come rapidly, or whether several tappings may be required with a possible chance of recovery. In the section on the general prognosis in portal cirrhosis the prognosis of ascites is referred to again (p. 295).

The **treatment** of ascites may be divided into: (i) paracentesis and drainage of the ascites; (ii) operations for producing vascular adhesions around the liver; (iii) diuretics and purgatives. The first and last of these methods are merely directed to the removal of the ascitic fluid. The production of artificial vascular adhesions is intended to prevent the peritoneal exudation, and is therefore a more radical method.

(i) *Advisability of performing Paracentesis.*—Formerly paracentesis of the abdomen was postponed as long as possible, partly because infection of the peritoneum sometimes followed tapping, and partly because the patient often went downhill extremely rapidly after the withdrawal of the ascitic effusion. With ~~antiseptic~~ precautions the likelihood of peritonitis resulting is minimised, while the occurrence of death comparatively shortly after paracentesis must be considered to be due to the natural course of the disease, and not to the removal of ascites.

Other reasons given for only adopting paracentesis as a last resource were that (*a*) the removal of fluid entailed the loss of a considerable amount of albumin, an argument that might also be used against opening an abscess (Murchison), and (*b*) that the intra-abdominal pressure of ascites prevented further transudation of fluid (Frerichs). Opinion has changed, and at the present time tapping is performed as soon as it is required in order to prevent the bad effects of excessive ascites.[2] It is a much more effective method than purgation, or the attempt to drain off the fluid by the kidneys, and does not tend to weaken the patient's strength as violent purging does.

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[1] Ramsbottom. *Med. Chronicle*, Manchester, 1906, xlv, 7.

[2] Austin Flint was the first vigorous advocate of tapping comparatively early in the course of the disease. Vide *Am. Journ. Med. Sc.*, 1863, xlv, 331; and *Brit. Med. Journ.*, 1883, ii, 565.

Bad Effects of Excessive Ascites.—The results of ascites have been already referred to; they are largely mechanical in the first instance and due to pressure on the abdominal and thoracic viscera; the functions of these organs are interfered with, and are therefore imperfectly performed. The renal veins are pressed upon and the urinary excretion impeded, much in the same way as in the backward pressure of heart disease.

Indications for Paracentesis.—When the patient complains of marked discomfort, the abdomen should be tapped; but apart from this there are other conditions, such as diminished amount of urine associated with oedema of the legs, signs of thoracic embarrassment, such as dulness at the bases of the lungs due to collapse, dyspnoea, râles at the bases of the lungs, and in rare instances haemoptysis from collapse of the lungs, which should make the practitioner tap the abdomen. In cases with considerable ascites in which haematemesis occurs, the abdomen should be tapped, as by this means venous engorgement may be diminished.

Method of performing Paracentesis.—The most satisfactory treatment is tapping the abdomen with a Southey's trocar and cannula, the fluid draining slowly away through a fine india-rubber tube into a bucket placed by or underneath the bed. Occasionally—I have seen three such cases—the abdominal wall is so enormously thick that a Southey's trocar fails to reach the peritoneal cavity, and a specially long trocar is required. In one such case in which I used the trocar of a Dieulafoy's aspirator the abdominal wall, as it became lax from the removal of the ascitic fluid, collapsed in thick folds on the trocar and bent it out of shape.

The skin should be carefully washed and an antiseptic dressing (1 in 40 carbolic) put over the area where the trocar is to be introduced, and the trocar and cannula should be boiled in a test-tube containing water and left to cool in a solution of carbolic 1 in 40. The trocar is usually inserted in the middle line between the symphysis pubis and the umbilicus, but sometimes, from the presence of omental or other adhesions, little or no fluid may be drawn off in this situation; in such instances it will be necessary to tap the abdomen in the linea semilunaris on one or other side. It has been suggested that the spot selected should be on a line joining the umbilicus and the anterior superior spine of the ilium on the left side, so as to avoid any danger of wounding the caecum or the liver (Plicque[1]). Puncture of the abdominal wall at the centre of this line has in at least two recorded cases caused fatal haemorrhage from a wound of the deep epigastric artery (Boidin,[2] Lian[3]). The middle line is therefore a safer site for the tapping. It is, of course, essential that the area selected should be dull, and it should be ascertained, if necessary by passing a catheter, that the dulness is not due to a distended bladder.

Under ordinary conditions and with reasonable care there should never be any danger of wounding the liver. I have, however, seen its effects at a necropsy, in which there was very considerable extravasation

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[1] Plicque. *Presse méd.*, Paris, 1900, p. 28.

[2] Boidin. *Bull. Soc. Anat.*, Paris, 1903, lxxviii, 415.

[3] Lian. *Ibid.*, 1907, lxxxii, 665.

from puncture of a large branch of the portal vein. As a rule, little or no harm results.

Goodhart,[1] indeed, refers to the case of a woman who, when apparently in a very advanced stage of the disease, was tapped, but only blood came away, and it was thought that the trocar had passed into the liver. However, from that time the patient steadily improved. Possibly this fortunate result was due to the wound producing vascular adhesions around the liver.

In very rare cases excessive haemorrhage may result from the trocar wound of the abdominal wall; this is likely to occur in patients in a haemorrhagic state.

A man aged forty-nine years, on the verge of a third attack of delirium tremens, had a number of subcutaneous haemorrhages, and had recently had profuse epistaxis. His abdomen was tapped in the middle line between the umbilicus and the symphysis pubis for ascites. When the cannula was withdrawn there was a spurt of arterial blood which was found to come from a branch of the deep epigastric in the substance of the rectus muscle; the artery was ligatured in two places, but the man died three hours afterwards. At the necropsy there were two pints of dark blood mixed with ascitic fluid in the peritoneal cavity; the liver (76 oz.) was remarkably cirrhotic, the veins of Sappey were dilated, and the body intensely anaemic. The blood appeared to have come from a wound of the deep epigastric veins, and to have passed through the track of the trocar into the abdomen. [St. George's Hospital P.M. Book, 1906, No. 371.]

The patient lies on his back, and the cannula should be fixed by means of plaster. During the drainage the abdomen should be compressed by a many-tailed bandage, or by a binder, which must be tightened from time to time, so as to avoid tympanites.[2] When the cannula has ceased to run from the small amount of fluid left in the abdomen, and not from displacement or blocking of the tube, which can be ascertained by passing a probe down the cannula, it should be withdrawn and the small wound covered with a plug of absorbent cotton-wool saturated with collodion. The binder should be kept on for two or three days to prevent flatulent distension of the intestines. Continuous strapping of the abdomen has indeed been recommended as a means of preventing the re-accumulation of fluid. If any distension occur, a dose or two of the sulphates of magnesium and sodium, a dram and a half of each, should be given. If the abdomen is nearly emptied, the small trocar wound heals up, and no trouble from leakage results. When, however, from adhesions the fluid is not satisfactorily removed, leakage from the wound may give considerable inconvenience, although the cannula has ceased to run. After paracentesis, from the increased absorption of fluid from the bowel and from the flaccid condition of the abdominal muscles, there is occasionally troublesome constipation.

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[1] Goodhart. *Guy's Hosp. Gaz.*, ~~June 7~~, 1897, 265.

[2] This practice appears to have been first employed by Dr. R. Mead [*Medical Works*, p. 518, 1762]; also see *The Gold-headed Cane*, p. 66, 1st ed., 1827.

Injection of adrenalin chloride into the peritoneal cavity after removal of the ascites has been followed by good results in cases thought to be cirrhosis; a dram of 1 in 1000 solution in ½ oz. of water may be used.

Use of a Large Trocar.—In former times a large trocar, like that employed for withdrawing the contents of an ovarian cyst, used to be plunged into the abdomen and the fluid rapidly drawn off. This is chiefly of historical interest; personally I have never seen it employed. The rapid withdrawal of fluid sometimes led to collapse, possibly from a large quantity of blood being drawn into the splanchnic veins.

Treatment by Continuous Drainage.—The wound has been kept open and the fluid allowed to run away continuously by Caillié, Elliot, Urso, Cheadle,[1] and others. But it has grave drawbacks.

Of Urso's 9 cases, 4 died directly; the best result seems to have been prolongation of life for nine months. Elliot's cases were both fatal. In some of Cheadle's cases in which the puncture remained open the results were satisfactory except for the manifest discomfort.

A modification of permanent drainage is described by Jaboulay[2] under the title "Cicatrice à filtration." In a man with ascites from cirrhosis the skin of the abdomen was incised transversely below the left costal arch, the muscles and peritoneum were divided lower down, and a piece of omentum was attached outside the peritoneal cavity, and the wound closed with hardly any escape of ascitic fluid. The skin healed over the artificially produced interstitial hernia of the great omentum, and the ascites diminished by escaping into the subcutaneous tissues of the abdominal wall. A week after the operation a small external fistula in the stitch wounds developed and the fluid gained an external exit. The man left the hospital in fair health. Narath in 1905 adopted a somewhat similar technique (*vide* p. 262). Paterson[3] drained the ascitic fluid into the subcutaneous tissues of the abdomen by means of a short glass tube with projecting flanges at each end. Permanent drainage into the tissues of the thigh through the femoral ring has also been employed (Wynter[4] and Handley). Routte's operation consists in making an anastomosis between the peritoneal cavity and the saphenous vein, and has been recommended in bad cases (Soyesima[5]). In 1911 Celso[6] collected 10 cases, only 2 of which were successful.

(ii) *The Surgical Treatment of Ascites by the Production of Vascular Peritoneal Adhesions.*—This method of treatment, as conceived by Drummond and Morison,[7] was based on the assumption that ascites was due to portal obstruction, and was an attempt to increase the collateral circulation between the portal vein and the general systemic veins. The operation was original in Morison's hands, but it had been previously planned by Talma and carried out by Van der Meulen in 1889, by Schelkley in 1891,

[1] Cheadle. *Brit. Med. Journ.*, 1900, i, 895.

[2] Jaboulay. *Lyon méd.*, 1900, xciv, 499.

[3] Paterson. *Lancet*, Lond., 1910, ii, 1273.

[4] Wynter. *Proc. Roy. Soc. Med.*, 1908, i (Clin. Sect.), 49.

[5] Soyesima. *Deutsche Ztschr. f. Chir.*, 1909, xcviii, 390.

[6] Celso. *Morgagni*, Milan, 1911, Riv., liii, 675.

[7] Drummond and Morison. *Brit. Med. Journ.*, 1896, ii, 728.

Autoserotherapy or injection into the abdominal wall of ascitic fluid has been followed by a urinary crisis & disappearance of the ascites. Vitry and Sézary injected 3 drams of ascitic fluid every other day.

Vitry et Sézary. *Rev de méd.*, Par., 1913, XXXIII, 86.

and by Lens in 1892. It is often called Talma's operation, or the Talma-Morison operation.

Technique of the Operation.—It is convenient to tap the abdomen before the operation so as to remove the ascites, though this is by no means essential. An incision parallel to the right costal margin is probably more convenient than one in the long axis of the body. The liver should be examined to confirm the diagnosis of cirrhosis, for the operation will do no good if the ascites is due to chronic peritonitis. The peritoneum over the liver and diaphragm is scraped so as to set up adhesive inflammation, and the surfaces are brought in contact by stitching the round ligament to the abdominal parietes or by passing stitches through the liver. The great omentum may be utilised to multiply the vascular adhesions by interpolating it between the diaphragm and the convexity of the liver. The numerous veins in the great omentum will assist in the development of a compensatory anastomosis. No bad effects, such as intestinal obstruction, result from limitation of the scope of the transverse colon's movement. In a case reported by Talma[1] the spleen remained of considerable size until at a second laparotomy the spleen was sutured to the abdominal parietes, after which it became smaller. It has therefore been thought that the peritoneum should be scraped widely so as to allow of the formation of vascular adhesions with the parietal peritoneum around the spleen and intestines (Ito and Omi[2]), as well as around the liver, but the production of widespread peritoneal adhesions is not advisable. The peritoneal cavity should be drained either by means of a glass tube inserted into the wound or by a separate suprapubic incision into the peritoneum so as to prevent the ascitic fluid separating the roughened peritoneal surfaces and interfering with the formation of vascular adhesions. The patient should also, as far as possible, be kept in a sitting posture, so as to prevent re-accumulation of fluid in the upper part of the abdomen.

It has been suggested by Murrell[3] that the development of a collateral circulation may be accelerated by rubbing the skin of the abdomen with ung. capsici or with cajeput oil. This would, of course, not be done until the operation wound had completely healed.

Epiplopexy is a slighter operation which requires less time and manipulation and is therefore applicable to advanced cases with little power of resistance. Its object is to increase the compensatory collateral circulation between the radicles of the portal veins and the systemic veins. The operation consists in suturing the great omentum to the anterior abdominal wall (Roberts[4]). In 1901 Schiassi[5] fixed the great omentum under the abdominal muscles and external to the parietal peritoneum; and in 1905 Narath[1] introduced the omentum into a subcutaneous pocket in the abdominal wall. These operations can be performed without general anaesthesia.

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[1] Talma. *Berlin. klin. Wochen.*, 1898, xxxv, 833.
[2] Ito und Omi. *Deutsche Ztschr. f. klin. Chir.*, 1902, lxii, 141.
[3] Murrell. *Lancet*, 1902, i, 1602.
[4] Roberts, J. B. *Phila. Med. Journ.*, 1901, vii, 163.
[5] Schiassi. *Semaine méd.*, Paris, 1901, xxi, 145.

Discussion of the Manner in which the Talma-Morison Operation improves the Condition of the Patients.—Does the improvement which sometimes results both in the general health and the disappearance of ascites depend solely on the increased collateral circulation and the resulting fall in the pressure in the portal vein? Against this interpretation it may be argued (*a*) that ascites does not occur when the blood-pressure in the portal vein is presumably highest—viz. early in the course of the disease when haematemesis is most often met with, and that experimental ligature of the portal vein does not necessarily produce ascites. Cirrhosis may exist for many years without ascites, which may appear quite suddenly; whereas if ascites were a purely mechanical effect of increased pressure in the portal vein, it should develop gradually and earlier in the course of the disease. It would therefore appear that the good effects of the collateral circulation are not solely due to relieving the pressure in the portal vein. (*b*) That ascites is a late manifestation and appears to be due to a toxaemic rather than a purely mechanical result of increased portal blood-pressure, and that it is caused by a poison in the blood exerting a lymphagogue action. The toxaemic state depends on hepatic insufficiency—in other words, on the inability of the cirrhotic liver to destroy poisons that are continually passing to it from the alimentary canal; these poisons, therefore, reach the general circulation and lead to oedema of the feet, ascites, and to constitutional and nervous symptoms. If the collateral circulation between the peripheral parts of the portal vein and general systemic veins is markedly increased, less blood will go through the liver and the toxaemia will be increased. In fact, the collateral circulation between the portal and general venous system is carried to its logical conclusion in Eck's fistula. In this experiment the portal vein is interrupted in the portal fissure; its proximal end is closed, and its distal end is put into communication with the inferior vena cava. In other words, the portal circulation through the liver is short-circuited and all the blood from the intestinal area enters directly into the inferior vena cava. As shewn by the experiments of Hahn, Massen, Nencki, and Pawlow[2] on dogs, this procedure tends to induce a disposition to uraemia. Thus, feeding the dogs on meat caused severe nervous disturbance, depression, asthenia, clonic and tonic spasms, and coma, and sometimes death. One of Morison's[3] patients was alternately excited and depressed for three weeks after the operation; similar depression has been noted in other cases, and may be explained as being due to the passage of poisons, manufactured in the intestine, directly into the general circulation without the intervention of the liver. Eck's fistula has been carried out on a man by Vidal,[4] the patient surviving for four months, ^

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[1] Narath. *Centralbl. f. Chir.*, 1905, xxxii, 433.

[2] Hahn, Massen, Nencki, und Pawlow. *Arch. f. exper. Path. u. Pharmak.*, 1893, xxxii, 161.

[3] Morison. *Lancet*, 1899, i, 1426.

[4] Vidal. *Semaine méd.*, Paris, 1903, xxiii, 351.

n), and on a woman by Rosenstein

ROSENSTEIN. *Arch. f. klin. Chir.*, Berlin, 1912, XCVIII, 843.

Thus, since it appears that the increase in the collateral circulation between the portal vein and the general systemic system would tend to induce a general toxaemia, the improvement in general health that follows the operation must be due to some cause other than the formation of anastomotic channels between the portal vein and the general systemic veins.

Thomson[1] suggested that the operation may prevent ascites by the simple method of producing universal adhesions and obliterating the peritoneal cavity. That this is possible is shewn by the cases, such as Dickinson's[2] and Weber's,[3] in which ascites disappeared and cirrhosis with universal peritoneal adhesions was found at the necropsy.

In 1899 Mr. G. R. Turner and I[4] suggested two other ways in which the development of a collateral circulation in adhesions around the liver might be beneficial to the economy: (1) By somewhat diminishing the flow of blood through the liver it may enable that organ to deal more satisfactorily with the blood passing through it, and so reduce the toxaemia, which is probably the important factor in inducing ascites. (2) That the presence of vascular adhesions over the surface of the liver would relieve venous engorgement and so allow a freer supply of arterial blood to the liver. The nutrition of the liver cells would thus be improved and they would be under better conditions to undergo compensatory hyperplasia. The compensatory hypertrophy of the liver[5] will enable the organ to perform more efficiently its important antitoxic functions, and so lead to a latency of the symptoms.

Early Performance of Operation Desirable.—It is important that the operation should be done comparatively early and not postponed until the patient is too debilitated to withstand it, for patients with cirrhosis are, at the best of times, far from good subjects. Thus patients may die from peritonitis or from shock immediately after the laparotomy. Another reason for not delaying operative interference is the importance of intervening before the liver tissue is so degenerated that it is unable to undergo compensatory hyperplasia as the result of the improved blood-supply provided by the adhesions. In suitable cases for operation medical measures should only be persisted in while the diagnosis is open to doubt, and they should always be directed to counteract any possible syphilitic disease of the liver. When medical treatment and a course of iodide of potassium have not benefited a case of ascites which is thought to be due to either syphilis or cirrhosis, the question of operative interference should be considered.

[1] Thomson, H. Campbell. *Med.-Chir. Trans.*, Lond., 1901, lxxxiv, 265.

[2] Dickinson, W. H. Allbutt's *System of Medicine*, 1898, v 691.

[3] Weber, F. P. *St. Barth. Hosp. Rep.*, 1898, xxxiv, 321.

[4] Rolleston and Turner. *Lancet*, Lond., 1898, ii, 1660.

[5] It is interesting to note that in a case of hepatic cirrhosis, in which it was suggested that the symptoms were arrested as the result of universal peritoneal adhesions, the liver weighed 89 ounces and presented nodules on its surface which may be interpreted as due to compensatory hyperplasia. The patient died from poisoning by mussels. (F. P. Weber. *St. Barth. Hosp. Rep.*, Lond., 1898, xxxiv, 321.)

When cirrhosis can be diagnosed with fair certainty in the pre-ascitic stage, and when there are evidences of the disease, such as haematemesis and splenic enlargement, operative interference has a much better chance than in the late stages.

The *contra-indications* to the performance of the operation are: (i) An advanced stage of the disease, shewn by debility, wasting, and marked toxaemia. (ii) Considerable jaundice. (iii) Definite heart or renal disease.

Results of the Operation.—A large number of cases have been operated upon, and in some instances the operation has been done on cases in which ascites was due to some other cause.

Sinclair White[1] collected 227 cases of cirrhosis operated upon, and found that 37 per cent were cured and 13 per cent improved, whilst in 15 per cent the operation failed, and in 33 death followed. At the French Surgical Congress in 1904 very divergent views were expressed: on the one hand, Monprofit[2] estimated that complete recovery followed in 35 per cent of 224 cases operated upon ; on the other hand, Willems[3] quoted statistics to the effect that only 10, or 4 per cent, of 250 cases operated upon were successful. In 288 instances in which the operation had been performed, about 70 per cent being cirrhosis, Bunge[4] estimated that cure occurred in 30 per cent, and improvement in a further 14 per cent.

On the whole, the results are somewhat disappointing, but this may in part be due to the fact that the operation is so often undertaken as a last resource, and late in the course of the disease, when the fatal termination is already near. The earlier the operation is undertaken, the better the chance of improvement or even arrest of the disease.

(iii) *Treatment by Diuretics.*—In moderate ascites diuretics may be tried; it is also advisable to give them directly after paracentesis has been performed. In considerable ascitic effusion, diuretics have little effect, possibly because the renal veins are pressed upon, and from the resulting venous engorgement the kidneys are placed at a disadvantage. For this reason they are often much more effective after paracentesis than before. Diuretics should never be employed to the exclusion of paracentesis. Many diuretics have been employed. A pill containing digitalis, squill, and mercury is one of the most popular diuretics in ascites. This pill is called Baillie's pill at St. George's, or Addison's pill at Guy's Hospital. Small doses of calomel may be given not only for their antiseptic action, but also to increase the output of urine, especially when combined with citrate of caffeine. Potassium salts, such as the acetate, citrate, and bitartrate, are recommended by some, but their diuretic effect is inferior to that of many other available drugs, and the depressing and toxic effect of potassium is a distinct drawback. It is better not to give spirituous solutions, such as spirit of juniper, spiritus aetheris nitrosi, as

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[1] White, Sinclair. *Brit. Med. Journ.*, 1906, ii, 1287.

[2] Monprofit. *Rev. de chir.*, 1904, xxiv, 602.

[3] Willems. *Ibid.*, 606.

[4] Bunge. *Die Talma-Drummondsche Operation*, 1905.

In Karell's cure, advocated in 1866, ~~consists in~~ the ~~patient~~'s diet ~~being~~ is restricted to 200 c.c of milk taken at 8. am, midday, 4 p.m and 8 p m and continued for 2 to 4 weeks after the fluid has disappeared (Goodman) it is possible they might, like alcoholic drinks, do further damage to the liver. Copaiba resin in 10 to 15 grain doses has been advocated as a valuable remedy in ascites. It should be given in keratin capsules, which resist the action of the gastric juice, so as to avoid irritation of the stomach. Apocynum is a powerful diuretic, as its name the "vegetable trocar" implies. It has the disadvantage of irritating the stomach; in order to prevent this, a small dose of cannabis indica should be combined with it. The tincture can be given in 15 to 30 minims three times daily, or the fluid extract (U.S.P.) in 10 minim doses may be employed instead. I have been disappointed with its effects in the ascites of cirrhosis. Asparagus has often been used as a diuretic. Hare[1] has seen improvement in ascites of cirrhosis after a dram of a liquid extract of asparagus given three times daily. Urea, on account of its diuretic action, has also been recommended. I have tried it without marked effect in several cases, and Goggi has seen bad results follow its administration. Diuretin has been tried, but is not of any particular use in the ascites of cirrhosis. Good results have been reported from nucleinic acid (Mezernitzky[2]). Liver substance or extract has been given by the mouth or by subcutaneous injection. Polyuria and diarrhoea result, and ascites may be much diminished. In 14 cases collected by Mouras[3] ascites disappeared in 7 (*vide* also p. 301).

Treatment by Purgatives.—The ancient treatment of attempting to remove ascites by free purgation has been abandoned, and vigorous drugs like elaterium and gamboge are no longer given. The excessive diarrhoea thus produced necessarily starves and weakens the patient, while it lessens the urinary excretion and so may tend to induce or increase retention of toxic substances in the blood and lead to toxaemia. Cheadle speaks of cases being "purged to death." Further, violent purgatives readily light up catarrh of the intestine, and so do harm. Repeated small doses of calomel may be given and exert a beneficial antiseptic action as well as clearing out the bowels. Saline purgatives such as sulphate of sodium or magnesium may be employed. Compound jalap powder in one-dram doses is an effectual purge. Turpeth, the Ipomoea turpethum of the Colonial and Indian Pharmacopoeias, is recommended in 20 grain doses (Murrell[4]).

Restriction of Fluid, etc.—Dickinson[5] records 6 cases of ascites due to cirrhosis treated by reducing the amount of liquid taken by the mouth down to one pint or less. In two cases the ascites disappeared. In one death occurred two years later from cerebral abscess, and the patient, a boy aged nine years, was found to have a hobnailed liver and universal peritoneal adhesions. The treatment was recommended not as a substitute for tapping, but as an adjuvant. The diminished fluid has distinct disadvantages, since it must tend to produce constipation, thereby increasing fermentation and autointoxication. Further, it must curtail the urinary excretion and so favour toxaemia.

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[1] Hare, H. A. *Therap. Gaz.*, 1899, xxiii, 589.
[2] Mezernitzky. *München. med. Wchnschr.*, 1910, lvii, 1032.
[3] Mouras. *Thèse de Paris*, 1901, No. 278.
[4] Murrell. *Lancet*, 1902, i, 1602.
[5] Dickinson. Allbutt's *System of Medicine*, 1898, v, 691.

It has been urged that the intake of chlorides should be restricted, as they are retained and tend to increase ascites,[1] but the results have not been very striking (Herringham and Hadfield [2]).

HAEMATEMESIS may be the earliest warning to the individual that he is the subject of any disease more serious than indigestion. A frequent sequence of events is that a man who for years has lived freely and has had attacks of gastritis and dyspepsia, due to alcoholic excess, has, while in his usual state of health or after a little uneasiness in the stomach or faintness, a copious haematemesis. Haematemesis may be preceded by abdominal discomfort, febrile disturbance, enlarged spleen, and evidence of gastritis. In such cases gastritis, due to toxic or infective agents, is probably the exciting cause of the haemorrhage. The haematemesis is sometimes repeated during the next few days, but usually there is a single large haematemesis. When haematemesis is often repeated the condition is either not cirrhosis, or, if there be hepatic cirrhosis, it is complicated by some factor, such as gastric ulcer, superficial gastric erosions, or ulcerated varicose oesophageal veins.

Incidence.—Haematemesis does not appear from statistics to be so frequent in the course of cirrhosis as is usually imagined. It is such a striking event that it is rather surprising to find that it occurs less often than jaundice and in about one-fourth of all patients dying with a cirrhotic liver.

In 243 cases, obtained by combining Yeld's[3] (85), Sears and Lord's[4] (78), and my own (80) cases, haematemesis occurred in 64, or 26·3 per cent.

21·7

It occurs rather more frequently in cases dying directly from the effects of cirrhosis.

34 26 24

In 80 cases of cirrhosis fatal from the effects of the disease, examined after death at St. George's Hospital, 26, or 32·5 per cent, had had haematemesis. In 4 of the 26 cases death was directly due to haematemesis. Men suffered more frequently than women; thus, of 51 men, 19, or 37 per cent, had haematemesis, whilst of the 29 women, only 7, or 24 per cent, had haematemesis.

34

Haematemesis is rare in children, possibly because alcoholism, which is certainly more frequent in adults, favours haemorrhage by reducing the coagulability of the blood.

Saunal[5] recorded fatal haemorrhage from an oesophageal varix in a girl aged twelve years with cirrhosis. In the Norfolk and Norwich Hospital Museum

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[1] Olmer et Audebort. *Rev. de méd.*, Paris, 1904, xxiv, 199.

~~[2] Herringham and Hadfield.~~ *St. Barth. Hosp. Rep.*, 1905, xli, 23.

[3] Yeld. *Ibid.*, 1898, xxxiv, 215.

[4] Sears and Lord. *Boston Med. and Surg. Journ.*, 1902, cxlvii, 285.

[5] Saunal. *Thèse de Paris*, 1892.

Restriction of chlorides' rarely affects ascites, but it may be of use in cases associated with oedema of the legs, in which after paracentesis the subcutaneous fluid is transfered to the peritoneum (Lemierre and LÉVESQUE)

LEMIERRE et LEVESQUE *Presse méd.*, Paris, 1922, XXX, 1029

Zelensky reported fatal
haematemesis in two
Jews aged 16 and 17 years
with cirrhosis.

ZELENSKY. *Ztschr. f.* l.c *Klin. Med.*, Berlin, 1912, LXXIV, 374 there is a specimen (No. 59) of dilated oesophageal veins from a girl aged eight years who died after a succession of haematemeses. The liver was hobnailed and weighed 36 ounces, the spleen weighed 18 ounces, the skin was slightly tinged, but there was no real jaundice.

After haematemesis the patient is left in a condition of secondary anaemia, the degree of which varies with the amount of blood lost. It is not easy to estimate the amount of blood lost, for though a considerable proportion of it may be vomited up, some always passes into the duodenum and gives rise to melaena, and may thus escape notice, and is necessarily difficult to estimate. After the haematemesis the patient sometimes feels relieved and loses any feeling of oppression and heaviness that he may have previously had in the abdomen. Enlargement of the spleen detected before haematemesis may diminish very greatly as the result of the loss of blood.

Although haematemesis is usually an early symptom, it may supervene late in the disease, and even prove fatal in a patient with ascites.[1]

A man aged fifty-four years was admitted into St. George's Hospital. He had jaundice, ascites, and albuminuria. Paracentesis to 10½ pints was performed, and after this the fluid did not collect again. He got weaker, and eventually died immediately after a large haematemesis. This was the only occasion on which he brought up blood. At the necropsy the liver was cirrhotic and weighed 6 pounds 10 ounces. The spleen, 10 ounces, the kidneys, 10 ounces each, appeared normal.

The blood is probably generally poured out slowly into the stomach, and thus has time to clot and to be acted upon by the gastric juice. It is for these reasons darker in colour than the blood in gastric ulcer, which is rapidly extravasated from the gastric arteries. The quantity of blood brought up is practically always considerable, for the vomiting is chiefly due to the mechanical distension of the stomach by the blood. When a small quantity is poured out the blood is not as a rule vomited, but passes into the bowel and gives rise to melaena. After a small haemorrhage the patient may vomit from some other cause, and bring up a little black or "coffee-ground" vomit, but this is somewhat exceptional. Very large amounts of blood may be vomited in the haematemesis due to cirrhosis. Thus Osler[2] refers to a case in which 10 pounds were lost in seven days. It is noteworthy that collapse is not so frequent or so marked as in the haematemesis of gastric ulcer, in which blood is rapidly poured out. When a varicose oesophageal vein is opened and very large quantities of blood are lost, sometimes at repeated intervals, death may follow, but this does not invalidate the general statement just made that in the ordinary haematemesis of cirrhosis alarming symptoms are rare.

After haematemesis the temperature is, as a rule, depressed from shock, but after a few days it becomes normal. It may then remain normal or become raised. Fever after haematemesis should suggest the presence of some complication, such as tuberculosis, a pleural effusion, especially on the right side, endocarditis, or an infective process in the tonsils, teeth, or elsewhere. Fever may, however, depend on rapidly progressing changes in the liver, especially when the spleen remains enlarged or increases in size. Haematemesis and rapid hepatic changes may be due to an infection falling on a liver in which cirrhosis was quiescent. In such cases ascites may soon appear.

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[1] H. Fagge. *Principles and Practice of Medicine*, ii, 134, 1st ed., 1886.

[2] Osler. *Practice of Medicine*, p. 558, 6th ed., 1905.

Possibly a slight and transient elevation of temperature after hæmatemesis may depend on constipation and the retention in the intestines of a considerable quantity of blood, and consequent microbic multiplication. Haematemesis may be such a severe shock to a patient who has been drinking steadily as to set up delirium tremens which may prove fatal.

Etiology of Haematemesis in Cirrhosis.—(1) *Gastritis.*—The haematemesis may follow a debauch and be due, in part at least, to an exacerbation of a chronic gastritis. In such a case the after-history may indicate whether acute gastritis alone or cirrhosis and gastritis together were responsible for the haemorrhage. Haematemesis apparently solely due to gastritis is by no means rare, but in any given case it must be borne in mind that haematemesis may after all have been disposed to by a cirrhotic liver, which subsequently became arrested and gave rise to no further symptoms.

Bad teeth and pyorrhoea alveolaris are not infrequently responsible for infective gastritis; and toxic gastritis may, of course, be set up by a drinking bout. In haematemesis from gastritis the blood is thought to be derived by oozing from the capillaries and small veins of the gastric mucous membrane. The haemorrhage is probably determined by minute areas of necrosis and ulceration in the course of a toxic or infective gastritis. When no source for haemorrhage can be found in the stomach or oesophagus it may depend on toxaemia, which shews itself elsewhere by haemorrhage from the gums, into the skin, etc.

It is unlikely that haematemesis is ever due solely to increase of venous blood-pressure in the walls of the stomach; some inflammatory or degenerate change in the gastric mucosa is a necessary factor. Often, of course, no determining cause is forthcoming; but sometimes fever, gastritis, and splenic enlargement may precede it; Gauthier[1] suggests that toxins unaltered by the liver damage the intestinal blood-vessels and so set up haemorrhage.

(2) *Minute Erosions.*—Haematemesis in cirrhosis may be due to superficial erosions of the gastric mucous membrane. These may be so small as to be easily overlooked, especially as the stomach is blood-stained and somewhat sodden. The stomach should be left for some hours in Müller's fluid, after which small erosions will become evident.

Deguy[2] reported fatal haematemesis in a case of latent cirrhosis with a minute superficial ulcer $\frac{8}{10}$ of a millimetre in size. Mathieu[3] recorded fatal

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[1] *Journ. de méd.*, March 10, 1896.

[2] Deguy. *Bull. Soc. anat.*, Paris, 1898, lxxiii, 767.

[3] Mathieu. *Semaine méd.*, 1897, xvii, 170.

haematemesis in a woman aged thirty who was thought to be the subject of gastric ulcer; the necropsy shewed cirrhosis, oesophageal varices, and numerous haemorrhagic erosions on the lesser curvature and posterior wall of the stomach. Microscopically there was acute interstitial gastritis, which was the apparent cause of the ulceration. In a man with enormously tortuous veins near the cardia fatal haemorrhage occurred from a small erosion; gastro-enterostomy had been done for a supposed ulcer. The liver was cirrhotic. The patient was a total abstainer (Taylor [1]).

(3) *Gastric and Duodenal Ulcers.*—Gastric ulcer rarely occurs in patients with cirrhosis of the liver.

100/ Among ~~[illegible]~~ cases of gastric ulcer examined after death at St. George's 3/ Hospital there were ~~[illegible]~~ with hepatic cirrhosis ~~(T. C. English)~~. According to the d/ Fenwicks [2] cirrhosis of the liver is present in 9 per cent of cases of gastric ulcer.

Duodenal Ulcer.—In rare instances, of which I have seen one, copious haematemesis may occur in the course of cirrhosis from a duodenal ulcer. In 184 cases of duodenal ulcer collected by G. M. Cullen,[3] hepatic cirrhosis was present in 2·2 per cent.

(4) *Varicose oesophageal veins* are the most important cause of haematemesis in cirrhosis, being present in the great majority (80 per cent) of the fatal cases in which a thorough necropsy is made (Preble [4]). Exceptionally varicose oesophageal veins are found when the liver is healthy.

Graham and Weir Mitchell [5] recorded cases of fatal haematemesis from this cause in a boy aged seventeen years and in a child with healthy livers. In adults oesophageal varix may be due to chronic alcoholism without any cirrhosis of the liver (Letulle [6]). It has been thought that this phlebectasis is due to the caustic action of alcohol on the mucous membrane of the oesophagus (G. Muller [7]).

This compensatory collateral circulation serves a useful end in relieving portal engorgement, but, like other compensatory mechanisms, it may fail. Severe haemorrhage may be due to ulceration of the mucous membrane covering the varices, and may be precipitated by the passage of rough masses of food through the oesophagus. These oesophageal varices and severe haemorrhages are commoner in association with large than with small cirrhotic livers.

The blood may well up from the oesophagus when the patient is lying quiet in bed, and not be vomited up as it is in gastric ulcer. But in this event the blood does not enter the stomach and will be alkaline. As the varicose oesophageal veins are practically always near the cardiac orifice, the blood usually runs into the stomach and is vomited as in ordinary haematemesis. An ulcerated varicose vein can be seen with the oesophagoscope (Abrand[1]). The loss of blood may be so profuse as to prove fatal.

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[1] Taylor. *Trans. Coll. Phys.*, Phila., 1906, xxviii, 17.
[2] Fenwick, S. and S. *Ulcer of the Stomach and Duodenum*, p. 76, 1900.
[3] Cullen. *Scot. Med. and Surg. Journ.*, 1897, i, 637.
[4] Preble. *Am. Journ. Med. Sc.*, 1900, cxix, 263.
[5] Graham and Weir Mitchell. *Trans. Assoc. Am. Phys.*, 1896, xi, 215.
[6] Letulle. *Bull. et mém. Soc. méd. des hôp. de Paris*, 1890, 3. s., vii, 783.
[7] Muller. *Gaz. hebd. de méd.*, Paris, 1900, v, 470.

It is highly probable that in many of the cases regarded as due to a general venous oozing, an ulcerated oesophageal varix was overlooked. The ulceration may be very small and may escape notice at the necropsy. Letulle says that the ulcer may only become apparent after the oesophagus has been left for twenty-four hours in Müller's fluid.

The first haemorrhage may be so profuse as to be immediately fatal. This was so in one-third of Preble's 60 fatal cases of gastro-intestinal haemorrhage in cirrhosis. In other cases repeated haemorrhages occur. In rare instances there are considerable intervals of good health between the attacks of haematemesis ; in Garland's[2] case haemorrhage took place at intervals for seven years. Repeated haemorrhages at brief intervals may kill the patient in a short time from the first symptom of illness (Marmasse[3]). In fatal gastro-intestinal haemorrhage from cirrhosis the primary disease is often latent. The latency of cirrhosis in cases with varicose oesophageal veins shews the value of this compensatory mechanism. In only 6 per cent of 35 cases of cirrhosis with varices were the clinical symptoms characteristic, viz. ascites, enlarged spleen, subcutaneous abdominal veins, etc. (Preble). The following case illustrates the latency of cirrhosis until fatal haematemesis from an ulcerated oesophageal varix occurs :

A man, aged thirty-three, was brought in dead into St. George's Hospital, having vomited up a large quantity of blood shortly before death. There were dilated varices at the lower end of the oesophagus, one of which was ulcerated. The veins at the cardiac end of the stomach were somewhat varicose and there was a very minute abrasion of the mucosa over one of them, but the vein, though exposed, was not perforated. The intestines contained blood. There was no ascites. The liver, 4½ pounds, was finely cirrhotic with bright yellow nodules. Microscopically there was multilobular fibrosis with much fatty degeneration of the liver cells. The spleen, 9½ oz., was shrunken as if it had been larger. The kidneys, 7½ oz. and 8½ oz., were hypertrophied but healthy. There was caseous tubercle at the apex of the right lung.

In the following instance death was due to haemorrhage from an ulcerated oesophageal vein in a patient under treatment for cirrhosis :

Charles W., fifty-one years, a farm labourer, was admitted under my care in St. George's Hospital on September 15, 1900, with slight ascites and oedema of the legs. He had been a moderate drinker, and had never had syphilis. In July 1900 he had haematemesis and melaena ; before this he had not suffered from dyspepsia or morning vomiting. He was a weather-beaten man with a large liver and slight ascites. The spleen was not palpable. There was oedema

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[1] Abrand. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1909, 3. s., xxvi, 378.

[2] Garland. *Trans. Assoc. Am. Phys.*, 1896, xi, 206.

[3] Marmasse. *Bull. Soc. Anat.*, Paris, 1899, lxxiv, 75.

of the feet, but no neuritis. Pulse 96, low tension, artery tortuous and somewhat atheromatous. Urine 1008, no albumin. The oedema of the feet went down and the man's condition improved ; he was indeed feeling particularly well, when on September 21 haematemesis occurred ; he was given suprarenal extract by the mouth and morphine hypodermically ; on the next day he again had haematemesis and became very blanched, so that concealed haemorrhage was suspected. He was transfused, but died the same day. At the necropsy there was a small ulcer in the oesophagus, elevated so as to look like a miniature volcano, situated about one inch from the cardiac orifice of the stomach. This opened into a vein ; the other veins at the lower end of the oesophagus were not varicose. The stomach and the intestines were full of blood. The liver, 80 oz., was pale, finely granular, and intensely cirrhotic, with numerous haemorrhagic spots in it. The portal vein was normal. The kidneys were pale but free from arteriosclerotic change. Spleen large. The cavity of the peritoneum contained two pints of ascitic fluid. Testes normal.

(5) *Varicose Gastric Veins.*—Haemorrhage from varicose veins in the stomach is very rare in cirrhosis. Letulle[1] described two fatal cases in men aged twenty-three and thirty-nine respectively. Other cases have been recorded by Marchiafava, Hillier, Blake, Jackson, Revillout, Minot, Taylor.[2]

Haemorrhage from the Pharynx.—Bouchard[3] pointed out that small angiomas are frequently present at the back of the pharynx and that haemorrhage from them may give rise to haematemesis. The pharynx of cirrhotic patients with haematemesis should therefore be carefully examined, as the cause of the haemorrhage, if found, can be satisfactorily treated by local styptics.

It has thus been seen that haematemesis in cirrhosis may occur (i) when there is no morbid lesion visible to the naked eye except gastritis. (ii) From small superficial abrasions of the gastric mucous membrane. (iii) In very rare instances from gastric or duodenal ulcer. (iv) From rupture or ulceration of varicose veins at the lower end of the oesophagus ; this is the most important cause. (v) From ulcerated varicose veins in the stomach. (vi) From the pharynx, the blood being swallowed and subsequently vomited.

Diagnosis of Haematemesis of Hepatic Cirrhosis from that due to other Causes.—Without mentioning all the possible causes of haematemesis it will be useful to refer to the causes which are most likely to be confused with hepatic cirrhosis.

(A) *Gastric ulcer* in men of mature years runs a very chronic course, gives rise to continued pain and dyspepsia, but very rarely goes on to perforation. In some cases that I have seen there was an absence of tenderness on palpation, although the ulcer, as verified at the necropsy, was large and adherent to adjacent parts. Haemorrhage from the stomach in these cases may give rise to a suspicion of cirrhosis. When a patient with entirely latent cirrhosis dies from very copious haematemesis, it is impossible to diagnose the condition from gastric ulcer except on the greater probability of cirrhosis giving rise to haematemesis in a middle-aged man than gastric ulcer.

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[1] Letulle. *Presse méd.*, Par., 1898, ii, 313.

[2] Taylor. *Trans. Coll. Phys.*, Phila., 1906, xxviii, 17.

[3] Bouchard. *Rev. de méd.*, Par., 1902, xxii, 837.

The occurrence of repeated haemorrhages and painful dyspepsia are against cirrhosis. Enlargement of the spleen militates against gastric ulcer. In young women gastric ulcer is far more frequent than cirrhosis. When cirrhosis occurs in them, there is usually considerable enlargement of the liver and spleen and evidence of alcoholic excess. Pressure over the gastric ulcer in young women causes sharp pain, compared by the patient to that of a knife, which is quite different from the diffuse tenderness elicited on deep pressure in gastritis accompanying cirrhosis. The blood in haematemesis due to cirrhosis is black from the prolonged stay in the stomach and is often clotted, forming masses that may stick in the patient's pharynx. In gastric ulcer the blood is poured out more rapidly from a leaking artery, and is therefore brighter, less acted upon by the gastric juice, and not necessarily coagulated.

(B) From *pore-like erosions of the gastric arteries.* Very free and repeated haemorrhages may take place from minute erosions. In these cases there is no deep tenderness and there is a comparative absence of antecedent gastric symptoms (Dieulafoy,[1] Steven[2]). The chief distinction from the haematemesis of cirrhosis seems to be the occurrence of repeated and copious haematemesis in a patient who has none of the signs, symptoms, or history of portal cirrhosis.

(C) In *gastric carcinoma* the vomited blood is black, resembles "coffee-grounds," and is usually small in quantity, so that melaena is not noticed. In carcinoma of the pylorus haematemesis is in rare instances profuse; in such cases the stomach will probably be dilated and a tumour may be palpable. As a rule, carcinoma of the stomach is accompanied by so much pain and the vomited blood is so small in amount that the condition is not likely to imitate cirrhosis. If tested, the vomit will probably be found not to contain hydrochloric acid. Another important point in the diagnosis of gastric carcinoma is the presence of a tumour near or at the umbilicus, in the line of the falciform ligament, or on the surface of the liver, or enlarged glands above the left clavicle.

(D) In *splenic anaemia* periodic haematemesis may occur for years; sometimes there is very fair health in between the attacks; in some instances recurrent haemorrhages have occurred over a period of ten years. The anaemia is more marked and the splenic enlargement more considerable than in cirrhosis. The diagnosis from cirrhosis, which depends largely on these points, is important inasmuch as splenectomy appears, from Osler's[3] and Harris and Herzog's[4] observations, to offer

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[1] Dieulafoy. (Exulceratio simplex.) *Presse méd.*, Par., 1898, i, 29.

[2] Steven, J. L. (Pore-like Erosions of the Gastric Arteries.) *Glasgow Med. Journ.*, 1899, li, 5.

[3] Osler. *Trans. Assoc. Am. Phys.*, 1902, xvii, 429.

[4] Harris and Herzog. *Ann. Surg.*, 1901, xxxiv, 111.

the best chance of cure. These cases usually occur in men between twenty and forty years of age, and so might easily be regarded as due to latent cirrhosis. Closely allied to chronic splenic anaemia of adults is the condition described by Banti,[1] and often spoken of as *Banti's disease*, in which splenic anaemia is after some years complicated by secondary cirrhosis of the liver.

In leukaemia, haematemesis and melaena may occur and be associated with both splenic and hepatic enlargement; the diagnosis from cirrhosis depends on an examination of the blood.

Prognosis.—Haematemesis is rarely fatal in cirrhosis; in ~~80~~ cases in which cirrhosis was the direct cause of death, haematemesis was the sole cause in only ~~4, or 5~~ per cent. 7

Treatment of Haematemesis from Cirrhosis.—The patient should be kept perfectly quiet in bed, and for two or four days nothing whatever should be given by the mouth. Ice should certainly not be sucked, as an indefinite quantity of water is thus taken, which necessarily sets up peristaltic contractions of the stomach, and so may give rise to fresh haematemesis, while, further, the cold water must abstract a considerable amount of heat from the patient, who is often already somewhat collapsed. Thirst, which is often a cause of distress to the patients, is a natural result of the loss of blood and of the draining of the tissues to replace the fluid part of the blood. The mouth should be cleaned by pads of moistened cotton-wool, and enemas of warm water should be given every four hours to relieve thirst. If necessary, subcutaneous transfusion with saline solution may be employed. In order to ensure rest a hypodermic injection of morphine is useful; this calms the natural mental disturbance and anxiety of a person who has unexpectedly brought up blood.

The administration of digitalis or digitalin, ergot, lead, or other drugs which constrict the vessels and raise blood-pressure, is useless if not dangerous. Nitrite of amyl, which dilates the small blood-vessels and lowers blood-pressure, has more in its favour.[2] Calcium salts increase the coagulating power of the blood, and so may lead to thrombosis in the bleeding vessels. A dose of a dram of the chloride may be added to one of the enemas of water given to relieve thirst, and may be repeated if haematemesis recurs and the patient's life is threatened from anaemia and exhaustion. As recurrent haematemesis in cirrhosis is usually due to ulcerated varices at the lower end of the oesophagus, it is advisable to try to act locally on the bleeding area by the administration of Ruspini's styptic, which is largely composed of gallic acid; the styptic may be given in half to one dram doses in an ounce of water by the mouth. Two successive half-dram doses may be given.

The various preparations of suprarenal gland substance may also be given by the mouth in recurrent haematemesis so as to obtain the local vaso-constrictive effect on the bleeding vessels. It should not be given hypodermically or per rectum, for, as its general effect is to raise the arterial blood-pressure, it would, if absorbed into the general circulation, tend to increase haemorrhage from any leaking vessel. Another remedy which may be given by the mouth is turpentine in twenty minim doses in spirit of chloroform (♏x.) and water (℥j.) every four or six hours.

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[1] Banti, G. *Semaine méd.*, Paris, 1894, xiv, 318.

[2] Compare Hare, F. *Lancet*, 1904, ii, 522.

The local application of an ice-bag to the abdomen has been recommended, but it may increase collapse. If there is great collapse after haematemesis, transfusion of normal saline solution subcutaneously or into the veins should be performed.

The lower bowel should be cleared out by a soap-and-water or by a glycerin enema, or when there is no recurrence of haemorrhage and there is any abdominal distension or slight fever, a blue pill followed by a saline draught containing sulphate of magnesium should be given on the third day. During the first three days it is better to give nothing by the mouth, rectal injections of water (10 to 20 ounces) being given to relieve thirst every four or six hours as seems necessary. Nutrient enemas or suppositories may also be given every six hours, but in many instances this is not really necessary, and the patient is more comfortable without. In fact, the mental influence of nutrient enemas is often more valuable than their physical effect.

If there is nothing to suggest gastric ulcer, cautious feeding by the mouth may usually be begun after the third day, provided there has been no return of haematemesis. Peptonised milk or peptonised milk gruel should first be given and the diet gradually improved. After a week or ten days, according to his general condition, the patient should be allowed to get up.

A most essential point is the after-treatment, which consists in a light, simple diet, abstinence from alcoholic stimulants and from highly spiced articles of food. The importance of change of life and of total abstinence should be clearly explained to the patient.

When, as sometimes happens, haematemesis occurs in patients with ascites, removal of the fluid by paracentesis may be followed by a cessation of haematemesis. In such cases congestion may be aggravated by the intra-abdominal pressure of ascites. Fatal gastro-intestinal haemorrhage has, however, occurred after paracentesis (Estachy[1]).

Haemorrhage from the bowel is common in cirrhosis. It may be divided into (i) melaena as ordinarily understood, viz. blood so altered that it is dark in colour; (ii) haemorrhage from piles or from the mucous membrane of the rectum, the blood being more or less of its normal colour.

Melaena (*ἡ μέλαινα νόσος*, the black disease) commonly accompanies or follows haematemesis. The blood, which, by distending, mechanically irritates the stomach, is expelled in two directions—into the oesophagus and into the duodenum. In other words, while haematemesis is going on the patient vomits into the duodenum. The haemoglobin of some of the blood-corpuscles is acted upon by the gastric juice and reduced to acid

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[1] Estachy. *Bull. et mém. Soc. méd. de Vaucluse,* Avignon, 1906, ii, 432.

In mesenteric thrombosis there is ^
more collapse and the constitutional
symptoms more severe than in
Melaena due to Cirrhosis.

haematin; there is hardly sufficient time for this to take place in the mass of the blood that is driven into the duodenum, so that the blackness and tarry appearance of the motions are largely the result of the iron of the haemoglobin being acted upon by sulphuretted hydrogen in the intestines and converted into some compound containing sulphide of iron.

Melaena from gastric haemorrhage may occur without concomitant haematemesis. When the volume of blood poured out is small, so that the stomach is not over-distended, it will pass into the duodenum mixed with the contents of the stomach and appear in the motions. In such cases the occurrence of melaena may escape observation or only be noticed accidentally, and may then be regarded as due to a latent duodenal or intestinal ulcer. It may be difficult or impossible to diagnose the source with certainty in the absence of other signs of cirrhosis. In duodenal ulcer there should be deep tenderness in the situation of the duodenum and pain two or three hours after food. Duodenal ulcer may complicate cirrhosis and give rise to melaena without haematemesis. I have seen one case in a man aged forty years in which this coincidence was present. The duodenal ulcer, undiagnosed during life, gave rise to very acute pain in the right loin.

Melaena in cirrhosis may depend on haemorrhages of considerable size from the mucosa of the intestines. It is usually seen about the same time as haematemesis, viz. in the pre-ascitic stage, but it may accompany considerable ascitic effusion, or occur after ascites has come and gone, and be a terminal phenomenon. Traces of blood are common in the ascitic stage (Crosa[1]); this occult blood, which cannot be detected by the naked eye, can be demonstrated by the delicate benzidine test.

A man aged fifty-two was admitted under my care with excessive ascites, which came on three months before, and melaena; his abdomen was tapped, but he rapidly sank and died within forty-eight hours of the paracentesis. At the necropsy there was cirrhosis, a complete absence of any dilatation of the oesophageal veins, a chronic gastric ulcer at the pylorus, and close to it a small ulcer of more recent date from which the blood filling the stomach and intestines appeared to have come. The portal vein was normal.

In the following cases extensive haemorrhage from the rectum immediately preceded death:

A woman of alcoholic habits, aged forty-nine, under my care in St. George's Hospital, was tapped for ascites twice within a fortnight after admission. After this she lived for seven and a half weeks in an extremely drowsy condition, but did not again become ascitic. The day before her death she passed 2 pints of blood from the bowel. There were no subcutaneous haemorrhages at any time. At the necropsy the mucous membrane of the lower 6 inches of the rectum was inflamed, and though no definite ulcer could be found, the rectum must have been the source of the haemorrhage; for no blood or blood staining was found higher up in the bowel. The liver was cirrhotic. In a man aged forty-four years death was immediately preceded by melaena. He had twice been tapped for ascites due to cirrhosis; he had successively haematuria, epistaxis, and lastly melaena.

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[1] Crosa. *Gazz. d. osp.*, Milano, 1909, xxx 1225.

Slight melaena may occur from small multiple haemorrhages from the mucosa of the intestines due to hepatic insufficiency. These extravasations are analogous to the multiple haemorrhage occurring under similar conditions into the skin and other tissues.

Piles occasionally cause some bleeding. It is remarkable that piles are not more frequent in cirrhosis, as it would naturally have been expected that portal obstruction would almost constantly lead to varicose haemorrhoidal veins. Frerichs,[1] Sappey, Thierfelder, Nothnagel,[2] and others are all agreed on the comparative rarity of piles in cirrhosis. It has been thought that haemorrhage from piles in cirrhosis serves a useful purpose by relieving hepatic engorgement, and that their cure by operation may be followed by haematemesis or by advance in the downward course of the disease; in other words, that piles in cirrhosis is a condition that it is dangerous to cure.

Treatment.—For considerable melaena without haematemesis a turpentine enema may be given. The patient should be kept quiet, if necessary, by morphine. If bleeding occurs from piles, the local condition should be attended to.

Haemoptysis in the course of cirrhosis is somewhat infrequent. When it does occur, it is usually comparatively slight. It may be due to various causes:—

1. *False Haemoptysis.*—This may depend on epistaxis from the posterior nares, or on haemorrhage from the mucous membrane of the naso-pharynx. These haemorrhages are not uncommon in the late stages, and may be due to the general toxaemia. Bouchard[3] insisted on the frequency of angiomas in the pharynx, from which haemorrhage may occur independently of grave toxaemia.

2. *Haemorrhage from the larynx* may be due to concomitant tuberculous disease, or to venous oozing depending on hepatic insufficiency. Dreyfuss observed a bleeding tumour in the larynx of a patient with cirrhosis.

3. *Haemorrhage from the lungs* due to (*a*) pulmonary tuberculosis; (*b*) extreme congestion accompanying pulmonary collapse secondary to abdominal distension; (*c*) venous oozing depending on hepatic insufficiency.

Haemoptysis in patients with cirrhosis should arouse a strong suspicion of pulmonary tuberculosis. It is, however, not always justified. In an alcoholic woman under my care who said she had had slight haemoptysis for years, the lungs were free from tuberculosis at the necropsy.

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[1] Frerichs. *Diseases of the Liver*, ii, 47. Transl. New Sydenham Soc., 1861.

[2] Nothnagel. *Diseases of the Intestines and Peritoneum*. English transl., p. 308, 1907.

[3] Bouchard. *Rev. de méd.*, Paris, 1902, xxii, 837.

[illegible] accom[illegible]

Bibliography.—Dreyfuss. *München. med. Wchnschr.*, 1898, xlv, 1022.—Galliard. *Union méd.*, Nov. 1889.—Lubet Barbon. *Ann. de laryngol. et d'otol.*, 1897 (Larynx).—Dieulafoy. *Manuel de interne path.*, ii, 689 (Larynx).—Debove et Trémolières. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1909, xxvi, 731 (Larynx).

Epistaxis is by no means infrequent in cirrhosis. It may be part of the general tendency to haemorrhage depending on hepatic insufficiency, but it may supervene before there is any other evidence of this toxaemia or be the first sign of this complication. It occurs from one nostril—usually, as was pointed out long ago, from the right. It usually comes from a point near the anterior extremity of the septum which can be easily compressed (Bouchard[1]). The haemorrhage may be due to ulceration of an arterial angioma, and may if uncontrolled be so severe as to lead to syncope. Relapses are very prone to occur. In a patient of Dieulafoy's[2] repeated haemorrhages occurred from an ulcerated angioma in the nostrils. Epistaxis may alternate with haemorrhages from other sources ([illegible]).

A man aged forty-four, under my care, had first haematuria, then epistaxis, and just before death melaena. His liver was markedly cirrhotic; he had been tapped twice for ascites.

Haemorrhages from other Situations.—Haematuria may occur from hepatic insufficiency, but it is rare.

In a man aged fifty years, who was under my care in 1903, with cirrhosis, transient haematuria occurred at the same time as epistaxis and oozing from the gums.

The following case is of interest as an example of haematuria and other forms of haemorrhage:

Cirrhosis. Haematuria due to Nephritis. Epistaxis. Fatal Melaena.—A man aged forty-four, of alcoholic habits, was admitted to St. George's Hospital under my care on June 20, 1900, with swelling of the abdomen, flatulence, pain near the umbilicus, swelling of the legs, and loss of flesh and strength dating from an attack of pneumonia eighteen months previously. He had the signs of cirrhosis with a large liver, but had very distinct haematuria with casts and a sp. gr. of 1009. The question arose whether there was cirrhosis with intercurrent nephritis or primary malignant disease of the kidney with secondary growths in the liver. There were no haemorrhages elsewhere. The abdomen required tapping twice. With rest in bed the haematuria passed away, but the patient's general condition got worse. On July 9 there was only a trace of albumin, and on July 11 none at all. On July 11 and 12 he had epistaxis, and on the latter day rapidly became comatose; though he revived a little after transfusion, he died on July 14 with considerable melaena. At the necropsy the liver, 68 ounces, was cirrhotic and finely granular; the portal vein was free from any thrombosis. The kidneys weighed $7\frac{1}{2}$ and 8 ounces each, and shewed parenchymatous nephritis. The mucous membrane of the small intestine was pigmented, the change being more marked near the stomach. The spleen weighed 10 ounces.

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[1] Bouchard. *Rev. de méd.*, Paris, 1902, xxii, 837.

[2] Dieulafoy. *Manuel d'interne pathologie*, 1901, ii, 669.

Menorrhagia and Metrorrhagia.—In the early stages of cirrhosis metrorrhagia is often seen; very possibly this is partly connected with chronic alcoholism. In the later stages of cirrhosis there is usually amenorrhoea, though when hepatic insufficiency supervenes, haemorrhages may occur from the uterine mucous membrane.

The occasional formation of a **haematoma in the rectus abdominis muscle** has already been referred to as a possible evil effect of the collateral circulation in connexion with the veins in the falciform ligament (*vide* p. 214).

In a case recorded by Lefas [1] in which portal cirrhosis was complicated by jaundice and grave toxaemia (icterus gravis) the cause of the haematoma was thought to be fatty degeneration of the endothelium of the intramuscular capillaries.

General Haemorrhages.—In the late stages of cirrhosis when the liver has become incapable of stopping poisons absorbed from the alimentary canal, a condition of hepatic toxaemia results. The blood shews a low fibrinogen content (Whipple [2]), the blood-vessels are damaged, and haemorrhages may occur all over the body. The general haemorrhagic condition may in some instances be due to haemic infection. Thus, in a case of cirrhosis with haemorrhagic bullae, Monnier [3] cultivated *Bacillus coli* and streptococci. In such cases there is fever. Petechial haemorrhages into the skin are frequent, and haemorrhages are often seen from the mucous membrane of the mouth, blood oozing from the gums or from cracks on the dry dorsum of the tongue. The sordes thus produced give rise to an offensive odour of the breath.

In an exceptional case recorded by Webber [4] haemorrhage took place from the external ears, in addition to haemoptysis and bleeding from the gums. Leudet [5] also reports haemorrhage from the external ear in cirrhosis.

Thrombosis and haemorrhages are usually associated with opposite conditions of the blood as regards coagulation, the blood coagulating more rapidly in thrombosis and less rapidly than normal in haemorrhage. In the following case they were associated:

An alcoholic woman aged forty years was admitted under my care for severe haematemesis thought to be due to cirrhosis; in two days' time she developed typical delirium tremens, from which she recovered under the administration of hyoscine $\frac{1}{75}$ gr. sub cutem. After her recovery from delirium she had numerous haemorrhages all over the body; it was noticeable that there were several small thrombosed veins in the neighbourhood of some large cutaneous haemorrhages. There were thrombosed veins on the front of the abdomen, on the right mamma, and right arm. The veins of the legs were not affected.

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[1] Lefas. *Bull. Soc. anat.*, Paris, 1902, lxxvii, 586.

[2] Whipple. *Arch. Int. Med.*, Chicago, 1912, ix, 390.

[3] Monnier. *Compt. rend. Soc. Biol.*, 1896, xlviii, 65.

[4] Webber. *Lancet*, 1894, i, 1000.

[5] Leudet. *Ann. mal. de l'oreille et du larynx*, Paris, 1890.

The *treatment* of haemorrhages is partly local and partly general; the local treatment consists in the application of remedies such as adrenalin to bleeding spots when they can be reached. The general treatment consists in giving lactate of calcium in half-dram doses three times a day for six doses, and in attempting to reduce the general toxaemia by diuretics such as citrate of caffeine, purgatives, and subcutaneous transfusion if necessary.

Nervous Symptoms.—Various nervous manifestations may occur in the course of cirrhosis; as a rule the more striking are noticed late in the disease and are associated with advanced toxaemia. The slighter nervous symptoms, such as headache, giddiness, muscular weakness and tremor, disturbance of mental power, disordered sensation, pruritus, may occur earlier in the course of cirrhosis. It may not be easy to determine satisfactorily in every case whether symptoms are solely due to the liver and its functional failure, to other factors, such as alcoholic excess or renal disease, or to hepatic inadequacy combined with one of these additional factors. Thus, delirium tremens is due to recent alcoholic excess, and other forms of delirium may be most satisfactorily explained as due to hepatic or renal inadequacy. As long as the kidneys excrete toxic bodies which the liver fails to stop, the patient may remain fairly free from nervous symptoms, but headache, mental torpor, and depression are readily produced by food which has no bad effects in a healthy person. Failure in the excretion of urine increases toxaemia and often precedes the onset of acute nervous phenomena.

Slight mental disturbance is common, but it is natural to ascribe loss of memory, failure of will-power and of intellectual ability, solely or in part to the effects of alcohol. As in other morbid conditions of the liver, mental depression and hypochondriasis are common. Muscular tremor is not infrequent, and is often largely alcoholic and due to sleeplessness or to nights disturbed by nightmares and bad dreams.

Pruritus.—Itching of the skin is generally associated with jaundice, but may occur in its absence; it is not a common symptom, and when it does occur is usually seen late in the course of the disease.

Hanot[1] speaks of a case in which it occurred, and was persistent, a year before symptoms of cirrhosis developed, without any jaundice.

The more severe nervous symptoms are coma, delirium, convulsions, and paralyses. It has been thought that a large cirrhotic liver is more prone to be associated with grave nervous symptoms, but there is probably not enough to justify this view.

Coma.—Cases of cirrhosis which run their full course usually die with coma, often when ascites has disappeared (post-ascitic stage) or is stationary and small in amount. Coma is due to grave toxaemia, which may be purely hepatic and depend on the destruction of the hepatic cells being so widespread that the liver fails to stop poisons manufactured in the alimentary canal, as a result of which the circulation becomes flooded with them. If the kidneys are active, the poisons may be got rid of, but in many cases coma is precipitated by failure in the excretory activity of the kidneys. Though coma is more commonly a terminal phenomenon, it may be transitory and be removed by treatment. I have several times seen coma disappear after intravenous transfusion; the same result may follow bleeding. A patient in a drowsy, semi-comatose condition may wake up sufficiently to try to get out of bed and then relapse into coma. Before the onset of coma there may be extreme irritability and restlessness, due to toxaemia and resembling that seen in uraemia.

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[1] Hanot, *Arch. gén. de méd.*, 1896, clxxvii, 67.

Delirium may be terminal and precede coma, or more rarely it may temporarily supervene in the course of the disease and disappear. In the case of transient delirium it must be borne in mind that delirium tremens is not uncommon in the course of cirrhosis. I have seen short bouts of fever associated with delirium lasting two days or so, recur in a patient with cirrhosis before ascites developed. The character of the delirium varies; usually it is low and wandering, but occasionally it is very violent. ~~The character of the delirium may change,~~ or quiet and noisy phases may alternate.

Delirium tremens may occur in various circumstances in the course of cirrhosis. It may come on in the usual way from continued drinking in a patient with latent cirrhosis, or even when the disease has advanced into the stage of ascites. It may follow haematemesis, and then be more of the nature of traumatic delirium[1] and comparable to the delirium which is precipitated, so to speak, by a fractured thigh or pneumonia in an alcoholic subject. In other cases in which haematemesis is not very profuse, a mild form of delirium tremens may develop and may possibly be due to the treatment—viz. deprivation of food by the mouth for a few days.

Convulsions are sometimes seen at the close of the disease. Delirium often passes into coma, and convulsions may be added shortly before death.

Paralysis.—In addition to paraplegia due to peripheral neuritis of alcoholic origin, other forms of paralysis occasionally complicate cirrhosis. Cerebral haemorrhage or thrombosis may supervene, but usually when the cirrhosis is latent. In very rare instances hemiplegia occurs without any gross change to account for it.

In an alcoholic woman aged fifty-three, under my care in St. George's Hospital, with cirrhosis, the onset of right hemiplegia and aphasia suggested cerebral haemorrhage. At the necropsy no naked-eye morbid change could be found in the brain. The liver was markedly cirrhotic (weight, 70 ounces). Lévi[2] records terminal coma and facial paralysis in a man aged seventy with an alcoholic history and cirrhosis, and without any cerebral change except oedema.

It is possible that cirrhosis might dispose to cerebral haemorrhage by producing a haemorrhagic tendency. I have seen a case of cirrhosis in a

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[1] Compare West, S. *Clin. Journ.*, 1895–6, vii, 58.

[2] Lévi. *Arch. gén. de méd.*, Paris, 1896, clxxviii, 165.

^/ But coma may appear rapidly in apparent perfect health (Cabot).

Cabot refers to 3 similar cases.

CABOT. *Journ. Amer. Med. Assoc.*, Chicago, 1912, LIX, 2295.

(HENRICI)

In an alcoholic woman aged 53 there were small haemorrhages in both lenticular nuclei and a large cirrhotic liver. This may be compared with the condition described ~~on p. 335~~ below.

HENRICI. *Lancet*, Lond., 1913, ii, 797.

girl aged nine years in whom there were multiple cerebral haemorrhages, fever, and probably a terminal infection. Haemorrhagic pachymeningitis may be associated with cirrhosis; Heitz,[1] who has collected 7 cases of these combined lesions, regards them as independent results of alcoholism. It has been thought that thrombosis in the cerebral vessels may be disposed to by hepatic cirrhosis.

In a girl aged twelve years hemiplegia was due to thrombosis of the cerebral veins. There was ascites due to hepatic cirrhosis, and it was thought by Fisher[2] that the thrombosis depended on absorption of organic bodies from the peritoneal cavity.

There is an interesting group of cases, in which cirrhosis is latent and the symptoms are entirely nervous. It is usually familial, but is not hereditary, and may be sporadic. Gowers[3] described it as "tetanoid chorea," S. A. K. Wilson[4] as "progressive lenticular degeneration." Wilson collected 11 cases, about equally divided between the two sexes and proving fatal in the second or third decade. A mixed cirrhosis of the liver and symmetrical degeneration in the lenticular nuclei are constantly found. ~~Homén[5] thought that the disease depended on hereditary syphilis; but Wilson, who excludes syphilis and alcohol, invokes a toxin probably not microbic~~ (for clinical features *vide* p. 335).

Duration.—Portal cirrhosis commonly lasts a considerable time; one two, or more years usually intervene between the appearance of symptoms due to the liver, if indeed their first appearance can be accurately determined, and the termination of the case. The progress of the lesion in the liver may become arrested, and from compensatory changes the disease may become latent. Sometimes, however, the disease runs a very rapid course; in such cases there are a certain amount of fever, abdominal pain on the right side, enlargement and tenderness of the liver, oedema of the legs, haemorrhages, and the early development of ascites. These cases are more often seen in comparatively young subjects, who have been drinking heavily, and may run their course in two to six months. This condition of acute cirrhosis is allied to subacute diffuse hepatitis (*vide* Symmers[6]). When cirrhosis runs a very rapid course, degenerative changes take place in the liver cells and the condition is much the same as icterus gravis, only less acute.

Termination and Method of Death.—As has been pointed out (p. 227), cirrhosis may be latent owing to compensation having been effected. The compensatory anastomosis of the oesophageal veins with the gastric veins may be a source of danger, and sometimes very profuse and even fatal haematemesis may result from ulceration or rupture of an oesophageal varix. In such cases cirrhosis may be quite unsuspected, although the fatal issue is a direct result of hepatic cirrhosis.

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[1] Heitz. *Rev. de méd.*, Paris, 1904, xxiv, 580.

[2] Fisher, T. *Lancet*, 1901, ii, 845.

[3] Gowers. *Rev. Neurol. and Psychiat.*, Edin., 1906, iv, 249.

[4] Wilson, S. A. K. *Brain*, Lond., 1912, xxxiv, 295.

~~[5] Homén. *Neurol. Centralbl.*, 1890, ix, 514.~~

[6] Symmers, D. *Am. Journ. Med. Sc.*, Phila., 1908, cxxxv, 251.

In only 6 per cent of 35 cases of haematemesis from oesophageal veins were the cases typical of cirrhosis from a clinical point of view (Preble[1]).

Haematemesis is rarely directly fatal. In ~~80~~ 96 cases fatal from the direct effects of cirrhosis examined at St. George's Hospital, ~~4, or 5~~ 7 per cent, only were due to haematemesis; but Preble has collected 60 cases of fatal gastro-intestinal haemorrhage in cirrhosis. Death may occur after a single large haemorrhage or after a series of repeated haemorrhages. In a third of the cases collected by Preble death followed a single haemorrhage.

Before the onset of ascites, death may be due to various complications (*vide* p. 283), which frequently throw any symptoms due to cirrhosis into the shade. Some of these complications are due to alcoholism; thus, there can be little doubt that alcoholism is in great measure responsible for the frequency of tuberculosis, peripheral neuritis, and cardiac failure. Generalised tuberculosis may carry off a patient with cirrhosis long before loss of flesh and impairment of general nutrition have appeared. I have several times seen generalised tuberculosis in fat patients with cirrhosis.

When chronic renal disease is associated with cirrhosis of the liver, the clinical aspect is mainly that of kidney disease, and death usually results from uraemia, which, it may be noted, is very much like the late toxaemic stage of cirrhosis.

Acute infections, such as erysipelas and pneumonia, when they occur in the subjects of cirrhosis, are extremely likely to prove fatal. It has been said that acute infections of this kind are more fatal in portal cirrhosis than in hypertrophic biliary cirrhosis, since the liver cells are in a better state of preservation in the latter disease. Occasionally acute infection may fall on the liver itself and set up acute degenerative changes in the liver cells, and so give rise to symptoms resembling those of acute yellow atrophy. But inasmuch as the liver is not healthy previous to the acute infection, as is the case in acute yellow atrophy, it is perhaps more convenient to speak of the condition as icterus gravis. An acute infection may attack the smaller bile-ducts without affecting the liver cells to such a degree as to produce acute yellow atrophy, though the clinical resemblance is considerable.

An alcoholic waiter, aged forty-seven, had had haematemesis seven weeks before death; ascites, jaundice, and oedema of the feet subsequently developed; he was admitted into St. George's Hospital and was tapped; he remained jaundiced, and for the last three weeks of his life was delirious. The liver, 60 oz., shewed multilobular and unilobular cirrhosis, with inflammation of the small bile-ducts, which contained inspissated masses of bile. The spleen weighed 10 oz. and was soft.

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[1] Preble. *Am. Journ. Med. Sc.*, 1900, cxix, 263.

When ascites has developed, death usually follows before the patient requires tapping more than twice. Indeed, if paracentesis has to be repeated several times, the case is either complicated with some degree of chronic peritonitis or is not one of cirrhosis at all. After one or two tappings the fluid may not reaccumulate and the patient may linger on for a time in a drowsy and very feeble condition—a post-ascitic stage. Death is usually due to increasing weakness and coma. There is often low, muttering delirium; active delirium is rare, but I have known it to be maniacal. More often the patient becomes more and more insensible and passes into deep coma. Occasionally while in this toxaemic state death may be precipitated by profuse haemorrhages from the stomach or rectum. Death may be due to some complication; in rare instances acute peritonitis results from infection by the trocar, but it occurs independently of tapping, and is then due to infection from within, either by the blood-stream or in rare instances from some lesion, such as tuberculous ulceration of the intestines, which allows micro-organisms to pass into the peritoneal cavity. Acute infections, such as pneumonia and erysipelas, may also prove fatal in a patient with ascites due to cirrhosis.

From a degenerated and fatty condition of the heart muscle sudden death may occur; this is by no means common in my experience; in 53 fatal cases of cirrhosis Cheadle[1] found that death occurred suddenly in this way in 6. More often a patient dies slowly with a failing heart but in a general toxaemic condition.

Complications.—**Tuberculosis.**—The liability of patients suffering from cirrhosis to be affected with tuberculosis of the lungs and peritoneum has been already referred to (*vide* p. 222). In about 12 to 14 per cent of cases with cirrhosis death is directly due to pulmonary tuberculosis; in many of these cases the cirrhosis is latent and is only discovered after death. In other instances pulmonary tuberculosis may escape detection or give rise to very few signs. Tuberculous peritonitis may not be suspected, as the ascites is very naturally regarded as due to cirrhosis. In tuberculous peritonitis supervening in cirrhosis there may be fever and more abdominal pain than in ordinary ascites, the onset of ascites may be very rapid, and if tapping be performed, the fluid is found of a higher specific gravity (1020) than in ordinary cirrhosis (1010); ~~cytological examination shews that~~ the predominating cells are lymphocytes instead of endothelial cells as in uncomplicated cirrhosis. (vide p. 247) In the following cases ascites was due to tuberculous peritonitis supervening in the course of cirrhosis:

A fat woman, aged forty-nine, with a marked alcoholic aspect, was admitted under my care at St. George's Hospital on January 9, 1902, with ascites, oedema of the feet, paraesthesia in the legs, and bronchitis. The liver was enlarged, the breath very foul, and there was morning sickness. She was treated as a case of cirrhosis, and on January 17 the abdomen was tapped;

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[1] Cheadle, W. B. *Some Cirrhoses of the Liver*, 1900, p. 51.

the fluid was turbid and of a specific gravity of 1020 ; owing to the thickness of the abdominal wall it was difficult to get a trocar sufficiently long to reach the peritoneal cavity, and only about a pint of fluid was withdrawn. Death occurred on January 19. At the necropsy the liver (68 oz.) was markedly cirrhotic and microscopically contained much fat ; the spleen weighed 12½ oz. Some chronic peritonitis and recent generalised tuberculous peritonitis, old tubercle at the apices and much bronchitis in the lower lobes of both lungs, were found.

A man, aged forty-seven, considered himself as well till September 7, 1908, when the abdomen suddenly became swollen. He was admitted into St. George's Hospital on September 14 with extensive ascites and was tapped the same day, 20 pints of fluid being removed. The predominating cells were lymphocytes ; there were a few endothelial cells and polymorphonuclear cells. He had a dry tongue, a persistently raised temperature between 100° and 102° F., and died after thirty hours of coma on September 30, twenty-three days after the onset. The necropsy shewed old cirrhosis, recent tuberculous peritonitis, and an old vomica at the apex of one lung.

Generalised Tuberculosis.—In cirrhosis the possibility of generalised tuberculosis should be thought of when there is continued fever and the patient rapidly goes downhill without any appreciable ascites. Generalised tuberculosis may prove fatal in fat patients.

Bronchitis.—Some degree of bronchitis is not uncommon and may be an accidental accompaniment. On the other hand, it may be directly related to the cirrhosis, and may be due to collapse of the bases of the lungs from upward displacement and impaired movement of the diaphragm due to ascites, flatulence, or an enlarged liver. It may be part of a general catarrh from alcoholic excess or be the result of backward pressure from a failing heart. Since what appears to be bronchitis may be really rapid pulmonary tuberculosis, it is advisable to test the sputum for tubercle bacilli in cases of bronchitis in cirrhosis. In the following case rapid tuberculosis gave rise to the signs of general bronchitis :

A fat alcoholic man, aged fifty, who had had delirium tremens, was under my care for a few days in St. George's Hospital in March 1900. He had oedema of the feet and a haemorrhagic eruption on the legs, a dilated heart, a large liver, and general bronchitis ; the sputum contained a little blood. The urine (1020) was free from albumin. He was thought to have cirrhosis of the liver and backward pressure from cardiac dilatation, with possibly a pulmonary apoplexy. After being in for three days, with delirium at night, he quite suddenly died. At the necropsy the liver (108 oz.) shewed multilobular cirrhosis with much fatty change ; the left lobe overlapped the spleen, which weighed 28 oz. The kidneys weighed 9 oz. each and were healthy. There was no ascites. There was a pint of clear fluid in each pleural cavity and very dense adhesions at both apices. There were small cavities at both apices and recent firm caseous masses in the lower lobes and miliary tubercles throughout both lungs. There was no pulmonary apoplexy. There was bronchitis and tuberculous ulceration of the larynx. The heart (17 oz.) shewed fatty infiltration and degeneration and was dilated, but was free from valvular disease. Examination of the lungs shewed tubercle bacilli.

Pleurisy with Effusion is not uncommon and is often a manifestation of concomitant pulmonary tuberculosis. It is more frequent on the right side, and this is very probably due, as suggested by Villani,[1] to a spread of inflammation or infection from the liver through the diaphragm. The pleurisy may be acute and accompanied by pain and fever, or of an indolent and chronic character. In the latter event the condition is much like a hydrothorax, but, as already mentioned, it is often tuberculous.

James [2] records a curious case of ascites and right pleural effusion, in which a communication was thought to exist between the right pleura and the peritoneal cavity, inasmuch as tapping the pleura emptied the abdomen, and, in fact, seemed to be more effectual than paracentesis abdominis.

The fluid may be serous, sero-fibrinous, or in rare cases haemorrhagic. Haemorrhagic pleurisy is usually due to tuberculosis (Barjon and Henry,[3] Jean [4]), though it is possible that in the late stages it may be due to hepatic insufficiency. It has been suggested that the haemorrhagic character of the pleural effusion is sometimes due to alcoholism (Fernet [5]). A pleural effusion, especially on the right side, is more likely to be haemorrhagic than a concomitant ascites. The following case illustrates the relationship of tuberculosis and a haemorrhagic pleural effusion:

A man, aged forty-nine, died in St. George's Hospital with cirrhosis of the liver and tuberculosis. At the necropsy there was a pleural effusion on both sides—serous on the right side, haemorrhagic on the left side; there were numerous tubercles in the lung on the side of the haemorrhagic effusion.

In the following case the haemorrhagic character of the pleural effusion probably depended on trauma:

A billiard marker, aged forty-three, was admitted under my care with fever, a large right-sided pleural effusion, enlargement of the liver, and ascites. The effusion was twice tapped, and clear fluid was drawn off on both occasions, which contained neither tubercle bacilli nor pneumococci. He passed into a toxaemic condition, and although transfused, died without any improvement. His abdomen never required tapping. The right pleura contained much dark blood-stained fluid; the lung was collapsed and covered with lymph, which was haemorrhagic in one place and suggested that the blood had come from a newly formed vessel; there was a little obsolete tubercle at the apex. There was serous ascites, no chronic peritonitis, a multilobular cirrhotic liver (65 oz.) with small bilirubin-calcium calculi in the gall-bladder. The round ligament contained a big vein the size of the little finger (*vide* Fig. 35).

Taylor [6] records a case of haemorrhagic pleural effusion which was independent of tuberculosis and probably due to an acute infection.

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[1] Villani. *Rif. med.*, Napoli, 1895, ix, pt. i., 686.
[2] James. *Trans. Med.-Chir. Soc.*, Edin., 1898–99, xviii, 191.
[3] Barjon et Henry. *Lyon méd.*, 1898, lxxxviii, 258.
[4] Jean. *Thèse de Paris*, 1891.
[5] Fernet. *Bull. Soc. méd. des hôp. de Paris*, 1900, 3. s., xvii, 781.
[6] Taylor, F. *Guy's Hosp. Rep.*, 1896, lii, 45.

The presence of a right-sided pleural effusion in a doubtful case is in favour of cirrhosis as against simple thrombosis of the portal vein, since it does not occur in the latter condition unless complicated by cirrhosis. It should, however, be borne in mind that some degree of dulness on percussion, loss of vocal vibrations and of breath sounds at the right base, may be due to upward projection of a large cirrhotic liver, or to a small cirrhotic liver pushed up by ascites, and not to any pleural effusion. In the same way friction at the right base may be due to local peritonitis and not to pleurisy.

Peripheral Neuritis.—The minor degrees of peripheral neuritis are often thrown into the shade by ascites or other effects of cirrhosis on which attention is focussed, and so pass undetected. Cramps, muscular tenderness, and loss of the tendo Achillis and knee jerks may occur in cases of cirrhosis admitted for ascites or haematemesis, and are, generally speaking, to be referred to alcoholism. Peripheral neuritis in the early stages of cirrhosis is usually alcoholic ^ ~~and may be associated with psychical symptoms, thus constituting~~ (Korsakoff's syndrome or polyneuritic psychosis). Well-marked alcoholic neuritis is commoner in women and is often associated with pulmonary tuberculosis; in such cases the liver is nearly always fatty and rarely cirrhotic, and the clinical picture is, in the main, that of neuritis.

In 114 cases in which the liver was cirrhotic at the necropsy there were 3 (females) cases in which death was due to alcoholic neuritis (Rolleston and Fenton). In 82 cases of cirrhotic livers at Charing Cross Hospital, there were 4 (2 males, 2 females) in which death was due to alcoholic neuritis (Candler[1]).

The terminal stages of cirrhosis are marked by symptoms due to hepatic insufficiency, such as epistaxis and other haemorrhages, delirium, and coma. The toxic coefficient of the urine will, if the kidneys be healthy, become increased as a result of the liver failing in its antitoxic function, and a toxaemic neuritis may result. If alcohol has been taken recently, the neuritis must be considered to be due to this cause, but in the absence of this factor hepatic insufficiency with resulting toxaemia is a satisfactory explanation. Very little alcohol may cause neuritis in advanced cirrhosis.

In a case of Gouget's[2] neuritis came on two weeks before death in a woman previously alcoholic, who, however, had had no stimulant for the two months that she had been in hospital. The arms and legs were simultaneously affected, which is unusual in the alcoholic form. Hayem observed the same type of neuritis in a case of primary carcinoma of the liver.

Delirium tremens has been dealt with on p. 280.

The occurrence of **chronic peritonitis** in the bodies of patients dying

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[1] Candler. *Arch. Neurol. Claybury*, 1907, iii, 441. The literature of polyneuritis in cirrhosis is given by Klippel and Lhermitte, *Semaine méd.*, Paris, 1908, xxviii, 13.

[2] Quoted in Lévi's *Paris Thèse*, 1896.

~~The neuritis~~ it may be due to a combination in varying degrees of alcoholism and hepatic insufficiency, and is said to be characterized by rapid extension to the arms, ~~incontinence~~ ~~of the~~ sphincteric disorder, and early onset of psychical disturbance (

with cirrhosis of the liver is referred to elsewhere (p. 223). The presence of chronic peritonitis in cirrhosis accounts for ascites, which lasts much longer than in cases of uncomplicated cirrhosis. As a result of chronic peritonitis the omentum may become rolled up and form a firm mass running across the abdomen.

In a case, recorded by Benham,[1] of cirrhosis complicated by chronic peritonitis the omentum and mesentery were thickened and matted together, and formed a pulsating mass during life.

Renal Disease.—The occurrence of albuminuria (*vide* p. 233), and the morbid lesions of the kidney (*vide* p. 224) in cirrhosis have been dealt with elsewhere. A priori it would naturally be expected that death would occur earlier in cirrhosis complicated by renal disease than in ordinary cirrhosis, inasmuch as the toxaemia depending on hepatic inadequacy would not be obviated by the excretion of toxic bodies by the kidneys. This hypothesis, however, is not supported by statistics. Both in Yeld's[2] figures from St. Bartholomew's Hospital and in the cases at St. George's Hospital the average age of patients dying with the combined conditions was higher than in those patients who succumbed to uncomplicated cirrhosis of the liver. When the two diseases are met with in the same person, the clinical symptoms are rather renal than hepatic. This is in accord with the hypothesis that in the combined condition the toxaemia would be more marked.

Gout not unnaturally figures in the history of some patients who come under observation with the symptoms of cirrhosis. Frank gout, however, is very rare in patients with active cirrhosis. A large liver, which may in some instances be due to early or latent cirrhosis, is not infrequent in gouty subjects.

In 43 cases of cirrhosis tabulated by Yeld the joints were healthy in 34 and contained uratic deposit in 9, or 21 per cent. In 5 of the 9 cases the kidneys were granular.

Terminal Infections are far from rare in cases of cirrhosis. Probably the bactericidal power of the blood is diminished as it is in chronic heart and kidney disease. Suppurative or fibrinous peritonitis is not a very uncommon termination; Garnier and Pignot[3] collected six examples of terminal pneumococcic peritonitis; streptococcic peritonitis may also occur; both of these forms may be independent of paracentesis. As already pointed out, tuberculous peritonitis may supervene in the course of cirrhosis.

Acute infection of the liver itself, giving rise to degeneration of the liver cells and to icterus gravis (Pitt,[4] Weber[5]), are probably not very infrequent. Erysipelas, pericarditis, pneumonia, infective endocarditis, are also occasionally met with.

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[1] Benham, F. L. *Trans. Clin. Soc.*, 1895, xxviii, 226.
[2] Yeld, R. A. *St. Barth. Hosp. Rep.*, 1898, xxxiv, 215.
[3] Garnier et Pignot. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1911, 3. s., xxxi, 469.
[4] Pitt, G. N. *Trans. Path. Soc.*, Lond., 1889, xl, 351.
[5] Weber, F. P. *Ibid.*, 1899, l, 136.

In 65 fatal cases of infective endocarditis tabulated by Kelynack[1] cirrhosis was found in 4. In the museum of St. Bartholomew's Hospital there is a spleen (2295 h) containing a mass of laminated blood-clot the size of an orange from a man who had cirrhosis of the liver and streptococcic endocarditis of the aortic valves.

In a case of cirrhosis of the liver Councilman[2] found infection with streptococci giving rise to purulent infiltration of the retroperitoneal glands, thrombosis of iliac veins and inferior vena cava, with secondary embolic infarcts in the lungs.

Thrombosis.—Thrombosis is rare in cirrhosis. Portal thrombosis when it occurs is frequently associated with cirrhosis, but it only occurred in 10, or 3·3 per cent, of 334 cases of cirrhosis examined after death at St. Bartholomew's Hospital (Langdon Brown[3]). I have seen severe phlebitis of the leg in the late stages of cirrhosis. A case of multiple thrombosis and haemorrhages was referred to on p. 278.

DIAGNOSIS.—Dyspepsia, often associated with evidences of alcoholic excess, of considerable duration, haematemesis which cannot be explained satisfactorily on other grounds, and enlargement of the liver and spleen, are the broad lines on which the disease can be diagnosed in the pre-ascitic stage. The onset of ascites in a patient who has manifested the above symptoms and is, in addition, more or less cachectic leaves little room for doubt that a late stage of cirrhosis has been reached.

The **differential diagnosis** of cirrhosis from other conditions has already been partially dealt with under the heads of ascites (p. 251) and haematemesis (p. 271). The diseases which simulate cirrhosis, in so far as the production of ascites and of gastro-intestinal haemorrhage are concerned, such as chronic peritonitis, in which is included perihepatitis, and gastric ulcer, have thus been considered.

From Hypertrophic Biliary Cirrhosis with Chronic Jaundice.—Portal and hypertrophic biliary cirrhosis, though distinct, may in certain instances be combined, and cases shew a transition from one to the other. It may be pointed out that the same blending of distinct types is seen in the case of kidney disease. Chronic parenchymatous nephritis, which may be compared to hypertrophic biliary cirrhosis, and the granular (arterio-sclerotic) kidney, which may be likened to portal cirrhosis, may overlap, run into each other, or be combined.

The clinical and anatomical features of the diseases may both be found in the same person; thus, in hypertrophic biliary cirrhosis a secondary portal cirrhosis frequently develops before death, and the patient, who has for years presented the symptoms and signs of biliary cirrhosis, dies with ascites and perhaps gastro-intestinal haemorrhage.

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[1] Kelynack. *Encyclopaedia Medica,* 1900, iv, 365.

[2] Councilman, W..T. *Trans. Assoc. Am. Phys.,* 1896, xi, 213.

[3] Brown, Langdon. *St. Barth. Hosp. Rep.,* 1901, xxxvii, 62.

Primary carcinoma arising in a cirrhotic liver is described on p.
Blumenau found that in 198 cases of cirrhosis carcinoma supervened in 3.5 per cent., or 10 times more frequently than in other conditions. It may occur in the cirrhosis of haemochromatosis (vide p.) and is said to be frequent in the cirrhosis due to schistosomiasis (PIRIE)

A correct diagnosis is not so easy as is often thought, especially in the early stages, and as has already been pointed out cirrhosis may be latent. In a series of 80 cases a correct diagnosis was made in 39 per cent (Cabot).

Blumenau. Arch. f. Verdauungskrankh., 1920, xxvii, 1.
CABOT. Journ. Amer. Med. Assoc., Chicago, 1912, LIX, 2295.
PIRIE Med. Journ. South Africa, 1921, xvii, 87.

Cases of portal cirrhosis with a large liver and rather persistent jaundice occasionally occur and very closely imitate hypertrophic biliary cirrhosis; the distinction between the two depends on the jaundice not being permanent, on the slighter degree of splenic enlargement, and on the occurrence of ascites or other characteristic evidences of portal cirrhosis. In cases of portal cirrhosis which run an acute course, or in which more acute inflammatory changes supervene on portal cirrhosis of some standing and affect the bile-ducts, the clinical and anatomical features may shew a combination of biliary and portal cirrhosis (compare p. 323).

From other Enlargements of the Liver.—A patient may come under observation, say for examination for life insurance or for digestive disturbance, and be found to have an enlarged liver without any other manifestation of cirrhosis. The question then arises whether there is latent cirrhosis or some other morbid change in the liver. If there is proof or history of alcoholic excess, the enlargement may be due to fatty change, to early or to latent cirrhosis, or to temporary engorgement. If the organ is smooth, free from tenderness and pain, and the spleen is not palpably enlarged, the probabilities are in favour of fatty enlargement. Tenderness is in favour of engorgement or of early cirrhosis; enlargement of the spleen points to cirrhosis or to malarial hepatitis.

Malarial Enlargement.—As a result of chronic and severe malaria very considerable enlargement of the liver with tenderness on palpation results. This is combined with splenic enlargement and usually with irregular fever. The patient is much run down in health and weight, and the condition presented is very like that of early but advancing cirrhosis. The history that the patient, often a young man and not specially alcoholic, has been the subject of continued malaria abroad is important. Examination of the blood is of course essential. These cases often improve very rapidly under treatment in England, and the enlargement of the liver is much diminished or disappears entirely.

Chronic Venous Engorgement.—The enlarged liver due to the backward pressure of obstructive heart or lung disease may imitate a cirrhotic liver; especially as there is not infrequently a transient systolic apical murmur in cirrhosis. In forming a diagnosis the history and appearance of the patient and the presence of other signs of cirrhosis or of heart disease are important. Thus alcoholism, haematemesis, and an enlarged spleen point to cirrhosis, while rheumatic fever in the past, haemoptysis, and beneficial results from digitalis or strophanthus, are in favour of chronic venous engorgement.

In *leukaemia* the liver is often much enlarged, and epistaxis and haematemesis may occur. In the myeloid form the spleen is greatly enlarged, while in the chronic lymph~~atic~~ form the superficial lymphatic oid glands are enlarged. The diagnosis is easily made by examination of the blood.

Malignant Tumours.—In the late stages when the patient is cachectic and ascites and jaundice are both present, the resemblance to malignant disease may be very close. In a patient over sixty years of age these signs are in favour of malignant disease. Enlarged glands above the clavicle are strongly in favour of carcinoma. A history of chronic alcoholism, long-continued dyspepsia, and splenic enlargement suggest cirrhosis. In many instances doubt must exist until the abdomen is tapped and the liver can be thoroughly palpated.[1] If it is small, it is probably cirrhotic; if large and smooth, it might be either cirrhosis or massive cancer growing inside the organ, but the former is much more probable, both because primary cancer is rarer and because it need not give rise to either ascites or jaundice. Enlargement of the liver with multiple umbilicated nodules points to malignant disease (*vide* also p. 520).

A *hydatid cyst* deeply embedded in the substance of the liver expands the affected lobe, makes it prominent, firm, and enlarged, usually in a downward direction. In cirrhosis the enlargement is more uniform, affecting both lobes, and the surface is slightly irregular. It must, however, be remembered that when a large hydatid destroys a considerable amount of liver substance, compensatory hyperplasia of the other lobe may occur, and that the resulting enlargement may appear uniform, as in cirrhosis. A large cirrhotic liver, unless displaced forward by a flatulent stomach, which is only a temporary condition, or by some other cause, is not nearly so prominent as in hydatid disease. In hydatid the general health is good, whereas in cirrhosis there is usually some impairment, or signs of chronic alcoholism.

Syphilis of the Liver.—A history of syphilitic infection, manifest lesions elsewhere, or a positive Wassermann reaction would point to this explanation of an enlarged liver. In syphilitic disease the liver is often irregularly enlarged, whereas the two lobes of the liver are generally uniformly affected in latent cirrhosis. Pain is more frequent in syphilitic disease, and albuminuria, if present, is of diagnostic value as pointing to the probable existence of lardaceous disease. Sometimes all external manifestations of syphilis are wanting and the only clinical evidence forthcoming is diminution of the enlarged liver under a tentative course of anti-syphilitic remedies, which should always be employed in a case of doubtful nature.

In *splenic anaemia*, in which recurrent haematemeses are frequent, the splenic enlargement and the anaemia are more marked than in cirrhosis, and there is usually a diminution in the number of white blood-corpuscles, or leucopenia. The diagnosis between splenic anaemia and portal cirrhosis may be rather difficult, since, on the one hand, splenic anaemia may terminate in cirrhosis of the liver (Banti's disease), and, on the other hand, both haematemesis and ascites may occur in cases of splenic anaemia in which fibrosis of the liver is absent, as shewn after death. Splenic anaemia with periodic haematemesis is chiefly distinguished from portal cirrhosis by the greater degree of anaemia and of splenic enlargement.

Kala Azar.—In countries where this disease is endemic, the diagnosis from cirrhosis must depend on the presence or absence of the Leishman-

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[1] Compare Landrieux. *Journ. des praticiens*, 1899, p. 737.

Donovan body. In Egypt there is a form of cirrhosis which must be distinguished from kala azar (Cummins [1]).

Intrahepatic Suppuration.—In acute cirrhosis accompanied by fever and considerable enlargement of the liver the diagnosis from intrahepatic suppuration might present difficulties. Haematemesis and an alcoholic history point to cirrhosis, while a hectic temperature, localised hepatic tenderness, leucocytosis, and a history of residence abroad and dysentery, or of conditions, such as recent appendicitis, suppurating piles, or gall-stones, which might lead to pylephlebitis or to suppurative cholangitis, are in favour of intrahepatic suppuration.

Carrington [2] described the case of a woman who had an enlarged liver and a temperature oscillating between 98·4° and 104°; pylephlebitis was diagnosed, but after death the liver weighed 72 ounces and shewed rapidly progressing cirrhosis and a fatty condition of the liver cells.

Enteric Fever.—In acute cirrhosis with a raised temperature there may be considerable resemblance to enteric fever. In patients who have been drinking heavily and present an enlarged liver and spleen, fever, and diarrhoea, a rapid diagnosis from enteric fever is sometimes only possible by means of a blood-culture or the agglutination test (Widal's reaction). In children with fever, diarrhoea, and swollen abdomen the suspicion of a perforating typhoid ulcer has even arisen (*vide* p. 220).

Intestinal obstruction has been suspected in a patient with repeated vomiting from alcoholic gastritis and abdominal distension from ascites. Considerable tympanites may come on rapidly with the onset of ascites. Careful examination of the patient's history and general condition should, however, correct a first impression of this kind and lead to appropriate treatment for the pressing gastric symptoms. Intestinal obstruction might conceivably occur from strangulation by a band of peritoneal adhesions in connexion with perihepatitis; and, of course, intestinal obstruction may occur in a patient with cirrhosis.

In Lusseau's [3] case of cirrhosis the third part of the duodenum was compressed by a cicatricial band.

PROGNOSIS depends in great measure on the reparative power of the body and on its ability to compensate for the lesions of cirrhosis. Some discussion on the compensatory mechanisms is therefore necessary in order to obtain a clear idea of the problems involved in the prognosis.

The organism attempts to compensate for the evil effects of cirrhosis in two ways: (i) By a further development of the normal anastomoses between the tributaries of the portal vein and the general systemic veins (*vide* p. 209). (ii) By hyperplasia of the liver cells.

(i) Dilatation of the communications between the tributaries of the portal vein and the general systemic veins compensates for the mechanical obstruction to the passage of portal blood through the cirrhotic liver. It thus relieves the engorgement of the portal area, and should thereby tend to obviate haematemesis, and probably to delay the onset of ascites and possibly to assist in its removal.

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[1] Cummins. "Kala Azar in the Anglo-Egyptian Sudan," *Third Report of Wellcome Research Lab., Gordon Memorial College, Khartoum*, 1908, p. 100.

[2] Carrington. *Guy's Hosp. Rep.*, 1883–4, series iii, xxvii, 337.

[3] Lusseau. *Progrès méd.*, Paris, 1879, vii, 545.

If the effects of hepatic cirrhosis were chiefly mechanical and due to venous engorgement of the portal area, the more extensively the portal vein was put into communication with the inferior vena cava, the better would be the result. But a most important element in the pathological results of cirrhosis is the destruction of the liver cells and the necessary loss of their various functions, especially their antitoxic action, or the power of stopping poisons absorbed from the alimentary canal and preventing their entry into the general circulation. If, therefore, this collateral circulation be carried to its logical extreme, namely, by making the portal vein open directly into the inferior vena cava, or Eck's fistula, so that the portal circulation is short-circuited and the liver is virtually put outside the circulation, the results, as obtained by Hahn, Massen, Nencki, and Pawlow[1] on dogs, are very striking. The symptoms were much like those of uraemia; the urine contained carbamic acid, which is normally changed by the liver into urea. Similar symptoms were produced by intravenous injection of carbamic acid, and hence it was regarded as the cause of the toxic manifestations.

It is therefore probable that the good effects of the collateral circulation in cirrhosis are not purely mechanical. Possibly diversion of some of the blood from the liver relieves congestion of that organ and so enables it to deal more satisfactorily with the remaining blood that still passes to it. It is also conceivable that the state of nutrition of the organ is improved by relieving the congestion of the portal vein, and that it is thus able not only to be more active functionally, but to undergo compensatory hyperplasia to greater effect. This subject was also dealt with in considering the way in which the Talma-Morison operation for the relief of ascites does good (*vide* p. 260).

(ii) The second anatomical change by which an attempt is made to compensate for the evil effects of cirrhosis is multiplication of the liver cells (*vide* p. 207). As a result of this hyperplasia the liver increases in size. This attempt at compensation in cirrhosis is very general, but its success is variable; in many instances it is in vain. Though for a time it staves off the evil day and the disease remains latent, there is always the danger that the cirrhotic process may spread to the hyperplastic areas and that they may become engulfed like the rest of the liver; further, the adenomatous nodules may outgrow their nutrition and become necrotic; or they may undergo fatty change from some general cause such as toxaemia, for example in the nodular cirrhosis sometimes seen in tuberculosis.

In some instances the compensation is sufficiently good to restore a

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[1] Hahn, Massen, Nencki, und Pawlow. *Arch. f. exper. Path. u. Pharmak.*, 1893, xxxii, 161.

fair equilibrium, and the disease is latent so long that it may be thought to be cured. There is hardly a return to perfect health, for there is a want of reserve power, and the compensation, if strained, may give way. The importance of this compensatory hyperplasia is well expressed in Hanot's dictum that the diagnosis of hepatic cirrhosis is made from the state of the connective tissues, and that the prognosis depends on the condition of the cells of the liver.

Period of the Disease.—In ordinary cirrhosis the prognosis is, generally speaking, gloomy, but there are several special points which must be taken into consideration. Thus the prognosis is necessarily different at an early and at a late period of the disease. It is quite possible for the earliest symptom of importance—haematemesis—to be succeeded by many years of life, and even for the cirrhosis, which gave rise to it, to remain permanently latent. But, though the disease may become latent, and remain so for years, the condition is not one of real cure. The morbid change in the liver, though compensated for, is still there, and may be restarted, or the compensatory mechanisms may fail. Thus, fatal haemorrhage may occur from dilated and varicose oesophageal veins, or, as just mentioned, fibrosis and fatty change may invade the areas of hyperplastic liver cells. The prognosis in the late stages is very bad, and ascites due to cirrhosis uncomplicated by chronic peritonitis is usually followed by death within a short time. A drowsy, sleepy condition points to a general toxaemia, and though it may be temporarily removed by treatment, it is prone to recur and pass into coma, and is therefore an extremely grave sign.

The **effect of treatment** has an important bearing on the prognosis from the point of view of preventing any further aggravation of the disease. Complete abstinence from alcohol is most essential, and on the patient's power of obedience his future will to a great extent depend. Marked improvement from iodides makes the prognosis good because it tends to alter the diagnosis of portal cirrhosis into that of syphilitic disease of the liver.

General Nutrition.—A well-nourished patient has naturally a much better prospect than an emaciated subject. The appearance of the face, haggard and sunken about the eyes and temporal fossae in cases rapidly going downhill, is an instinctively recognised guide in forming a very grave prognosis, while the nutrition of the skin elsewhere, and of the muscular development generally, is of considerable value. The presence of stigmata on the face has been said to be of bad prognosis, but as an isolated manifestation this has seemed to me of little value; a dirty, earthy tint of the skin is more significant of a general nutritional change.

Age has some influence on the prognosis. Thus, Cheadle[1] found that the average age of 37 cases in which temporary or prolonged improvement occurred was thirty-nine years, or nearly ten years less than the average age of fatal cases of cirrhosis. It is possible that in these cases the cirrhosis developed rapidly as the result of heavy drinking, and that the process was not so advanced as in older subjects, in whom the fibrosis is formed more slowly. A comparatively early age is more favourable, inasmuch as the general nutrition is probably fairly preserved, and reparative and compensatory processes can be carried out efficiently. On the other hand, the prognosis in children is bad.

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[1] Cheadle. *Some Cirrhoses of the Liver*, p. 72, 1900.

Size of the Liver.—From what has been said about compensatory hyperplasia it would appear probable that enlargement of the liver improves the prognosis, since it is more likely to be associated with latency of the disease than a small one. But this is not a hard-and-fast rule, since enlargement may be temporary and due to alcoholic excess, to absorption of poisonous products from the alimentary canal, or to complications such as cardiac failure and backward venous pressure. Further, in the early stage when the cirrhotic process is actively advancing, the liver is considerably enlarged. But in the absence of constitutional symptoms, complications, or other signs that the cirrhosis is active and progressive, the outlook is better in a case with a large than with a small liver. This is borne out by the fact (*vide* p. 197) that the liver is larger in persons affected with cirrhosis but dying from accident or from independent diseases than in patients dying directly from the disease. Most of the cases of cirrhosis reported as recoveries have enlarged livers. According to Hanot and Gilbert[1] and Cheadle, two-thirds of the cases conform to this statement. Cheadle dealt with no less than 30 cases in which recovery, either temporary or prolonged, resulted after tapping.

Enlargement of the spleen is present in progressive cases, and is absent, as a rule, in latent cases in which compensation has taken place. Its size is, therefore, an indication of some value, and in cases thought to be latent, splenic enlargement should suggest the possibility of approaching haematemesis, ascites, or toxaemia. It should be regarded as a danger-signal and as an indication for purgation by which portal engorgement and intestinal fermentation can be diminished or obviated. If the spleen remain enlarged after haematemesis, the possibility of a recurrence or of the near advent of ascites should be borne in mind.

Haematemesis is usually an early symptom; often, indeed, the first indication to the patient that ~~he has~~ anything serious ‸ the matter. In ‸| is such cases the immediate prognosis is usually good, for the patient recovers, and may by a careful life escape any further manifestations of cirrhosis for years, or possibly for ever. Haematemesis is rarely fatal, but when it is so, the fatal haematemesis is usually the first. Haematemesis frequently repeated at short intervals is probably due to an ulcerated oesophageal vein, and the prognosis is worse than in the ordinary single haematemesis. When haematemesis occurs in patients with ascites, the prognosis is even more gloomy than it was before, as its occurrence makes it probable that the ascites is due to cirrhosis of the liver and not to concomitant chronic peritonitis.

Multiple or general haemorrhages, from the gums, throat, nose, skin,

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[1] Hanot et Gilbert. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1890, 3. s., vii, 492.

SEX. men are more prone to ascites and death from cachexia, women to show larger livers and death from hepatic insufficiency (Chauffard ~~[illegible]~~ and Brodin ~~[illegible]~~)

Chauffard, A. et Brodin, P. Bull. Acad. de méd., Paris, 1924, 3e ser., xci, 569

Weber reported Cases in which
ascites was tapped and disappeared,
examination after death some years
later, in one instance 8½ years
shewing compensatory circulation in
adhesions presumably set up by
the paracentesis

Weber, F. P. Proc. Roy Soc. Med., 1924, XVII
(clin. Sect):

etc., make the prognosis very grave, as they point to an advanced toxaemia, which may be succeeded by coma.

With the onset of **ascites** the patient's days are, as a rule, numbered. According to Hale White,[1] indeed, cases of uncomplicated cirrhosis, *i.e.* without chronic peritonitis, never survive to be tapped more than once. Though this may not be strictly in accord with general impression, and though recovery after paracentesis has often occurred in cases in which the existence of cirrhosis has subsequently been established after death from other causes, the gravity of ascites due to cirrhosis can hardly be overestimated.

But the onset of ascites in a patient thought to have cirrhosis must not be regarded as necessarily the equivalent of a death warrant. Many recoveries after paracentesis for ascites thought to be due to cirrhosis have been recorded (Bristowe,[2] Millard,[3] and Cheadle [4]).

An instructive case is mentioned by Hawkins [5] of a man who had been a hard drinker and was under Murchison in St. Thomas's Hospital with ascites. After this he became a total abstainer and lived in fair health for twelve years. At his death from pericarditis and granular kidneys, the liver was found to weigh 59 ounces, its capsule was much thickened and adherent to the diaphragm, and on section it shewed multilobular cirrhosis. Ehret [6] reports the details of a man who thirty years before his death, which was due to carcinoma of the oesophagus at the age of seventy-two, had jaundice, ascites requiring several tappings, and pain over the liver and spleen ; he became temperate. The liver was small and cirrhotic, the spleen large and surrounded by firm adhesions.

When, as in Hawkins' case, death subsequently occurs from some other cause, and the diagnosis of cirrhosis is confirmed, we must conclude that the compensatory mechanism is sufficient to explain the cure. It must be remembered that ascites associated with cirrhosis may be due to some concomitant cause, such as chronic peritonitis or mitral disease. In such cases death is less likely to supervene rapidly than in cases in which there is no cause for ascites other than cirrhosis. In ascites due to chronic peritonitis repeated tappings may be required, and the outlook as regards prolongation of life is better than in ascites due solely to cirrhosis.

If in a case thought to be cirrhosis the ascitic effusion is loculated from the presence of adhesions, it is probable that for this reason the prognosis is better than in cases in which there are no grounds to suspect adhesions. But, on the other hand, it is difficult to be certain that cases of loculated or encysted ascites are not entirely due to chronic peritonitis, to the exclusion of cirrhosis.

Oedema of the legs is, apart from any local cause, of bad omen, for it either points to a general toxaemia, cardiac debility, or is associated with, and possibly due to, pressure of the ascitic exudation on the inferior vena cava. In these conditions the outlook is extremely bad.

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[1] Hale White. *Guy's Hosp. Rep.*, 1892, xlix, 1.
[2] Bristowe. *Trans. Med. Soc.*, 1892, xv, 271.
[3] Millard. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1892, 3. s., ix, 153.
[4] Cheadle. *Some Cirrhoses of the Liver*, p. 69, 1900.
[5] Hawkins, H. P. Allbutt's *System of Medicine*, 1897, iv, 180.
[6] Ehret. *München. med. Wchnschr.*, 1903, xxxix, 321.

Functional activity of the kidneys, or renal permeability, is an important factor in the prognosis, and the urine should always be measured and examined, so that any feature in its amount and in its solid contents may be at once detected and treated. So long as the kidneys remove the toxic bodies which the cirrhotic liver allows to pass into the general circulation, the patient is in a fairly satisfactory state ; but failure in the urinary excretion brings on a condition of hepatic toxaemia which resembles uraemia. The presence of diacetic acid, as shewn by a port-wine colour on the addition of ferric chloride, in the urine makes the prognosis very grave, as acid intoxication is imminent and may lead to coma.

Fever is met with in cases which run a comparatively acute course, or in the presence of some complication such as tuberculosis, and is therefore of bad prognosis.

Complications, such as pulmonary tuberculosis and peripheral neuritis, make the prognosis graver. When a granular kidney is associated with cirrhosis, the age at death is higher than in cases fatal from uncomplicated cirrhosis, and clinically the renal symptoms are more prominent. It need hardly be added that any acute infection, such as peritonitis, pericarditis, erysipelas, or pneumonia, necessarily makes the outlook extremely bad.

The integrity of the hepatic cells is of great importance in recovery from erysipelas (Roger and Garnier[1]). In ordinary cirrhosis in which the cells are much affected erysipelas is usually fatal. Ten cases of ordinary cirrhosis attacked with erysipelas all terminated fatally (Bridiers de Villemor[2]), whereas in hypertrophic biliary cirrhosis, in which the liver cells may remain intact and actively functional, recovery may be hoped for.

TREATMENT will be considered under the following heads :—

1. To prevent any further advance in the morbid change in the liver. This includes abstinence from alcohol, careful dieting, and special attention to the alimentary canal so as to prevent or minimise autointoxication. In the early pre-ascitic stage of cirrhosis these measures may be spoken of as prophylactic.

2. The palliative or symptomatic treatment. The treatment of conditions such as haematemesis (p. 273) and ascites (p. 257) will be found under those headings.

3. To promote the compensatory mechanisms by means of which the disease becomes latent.

1. **To Prevent and Remove Factors which favour the Morbid Change in the Liver.**—Irritating or toxic substances, such as alcoholic drinks and spicy or stimulating food, must be avoided. Alcohol must be entirely prohibited, and care should be taken not to give the patient

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[1] Roger et Garnier. *Rev. de méd.*, Paris, 1901, xxi, 97.

[2] Bridiers de Villemor. *Thèse de Paris*, 1893-94, No. 428.

When oedema of the feet precedes
ascites the course of the disease
is likely to be rapid (Vide Presle)

Presle. These de Paris, 1891-92

alcohol in his medicines. Thus, tinctures, alcoholic extracts and spirituous solutions, such as spiritus chloroformi, should be avoided, and infusions or watery extracts substituted. If it is impossible to prevent the patient taking some stimulant—a relaxation of his strict duty to the patient—the medical attendant should see that it is taken after meals and largely diluted. On a patient's power of will to become a total abstainer his future will largely depend. It may be very difficult, from the nature of their occupation, for some persons, such as those engaged in the liquor traffic and commercial travellers, to become teetotallers. In such instances change of occupation or retirement from business is advisable.

Most writers agree that in the last stages of the disease alcohol is necessary; according to Cheadle,[1] indeed, total deprivation of alcohol hastens the final collapse. The administration of alcohol in the very last stage, when death seems imminent in a few days, is really only a kindly act and directed to inducing euthanasia. Any attempt at curative treatment is over, and the passing of the bar is thus made easier.

Diet is a most important part of the treatment. The object is to minimise autointoxication and to prevent further progress of the cirrhotic process. Milk is the ideal diet; as the fat in it is well emulsified absorption takes place, even under the difficulties presented by intestinal catarrh. The protein of milk is much less harmful than the proteins of meat, since the putrefactive products derived from it are minimised and the liver is less taxed in the production of urea. Milk has some diuretic action on the kidneys and leaves little residue, so that gastro-intestinal autointoxication is much reduced. A patient should be put on milk diet and kept on it till his condition has markedly improved. Difficulty will often arise in getting the patient to carry out the strict diet, from its monotony. Four pints of milk may be given during the twenty-four hours, or a larger quantity of skimmed milk. Milk may be diluted with mineral waters, such as soda, Apollinaris, Vichy, Vals. A little bicarbonate of sodium may be added with advantage and may do good by antagonising acidosis. Milk may be given as junket, in the form of milk jelly, or flavoured with a little tea, coffee, or cocoa, or as a soup with a vegetable flavouring. Milk should be given every two or two and a half hours during the waking hours. When digestion is defective, milk should be peptonised or given in the form of Benger's food. In some persons milk gives rise to nausea. Herter[2] finds that this can be obviated by substituting skimmed milk. The percentage of fat is thus reduced from 4 to 1 per cent. Kumiss, or fermented mare's milk, may also be given when ordinary milk disagrees, but should not be continued longer than necessary. It is very readily absorbed, and may be given in much larger quantities than ordinary milk. Whitla[3] recommends buttermilk, or the kumiss made in Ireland by mixing half a pint of water, half a pint of buttermilk, and four pints of fresh milk, and one ounce of loaf sugar, leaving in a warm place for thirty-six hours and occasionally shaking. A trustworthy form of soured milk may be tried.

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[1] Cheadle. *Brit. Med. Journ.*, 1900, ii, 693.
[2] Herter, C. A. *Lectures on Chemical Pathology*, p. 88, 1902.
[3] Whitla, W. *Dictionary of Treatment*, p. 502, 3rd ed., 1896.

It has been said that a restricted milk diet is well borne by French and Italian, but not by German, patients. A milk diet should be maintained until improvement occurs; eggs and some easily digested farinaceous and protein foods should be added to the diet, and if these are well borne, pounded fish should be given after an interval. Beef-tea, meat essences, strong soups, and butcher's meat are not advisable. As a change, fruit and vegetables may be occasionally allowed. Stimulating and spicy food, such as curries, pickles, anchovies, pickled fish, vinegar, ginger, should be avoided. Fatty food, inasmuch as by fermentation it may give rise to fatty acids, such as acetic, lactic, butyric, valerianic, should be avoided. Coffee and tea should be taken in small quantities freely diluted with milk.

Carbohydrate Food.—The glycogenic function of the liver is well preserved in spite of the marked histological changes in the organ, and theoretically sugar and starchy foods should be readily assimilated. In practice carbohydrate food should be given in very small quantities and only increased if it is well borne. From the frequency of gastro-intestinal catarrh, fermentation is very likely to take place in sugary food and to give rise to dyspepsia, flatulence, and the production of acids, which when absorbed may further degenerative and cirrhotic changes in the liver.

A grape cure, as much as 5 lbs. of ripe grapes daily, has been employed with some success by Cavazzini,[1] who ascribes good results to the sugar and the ferments in the grape juice.

Drugs.—Iodide of potassium should always be tried in cases thought to be cirrhosis of the liver, ~~on the chance that the condition is in reality syphilitic~~. Whether iodides do any good in ordinary cirrhosis is very problematical, and no reliance can be placed on this drug or on chloride of ammonium, though it is well to give them a trial. Cases of early cirrhosis often improve under chloride of ammonium, but the other hygienic measures adopted may in reality have been responsible for the good effects ascribed to that drug. Iodoform has been employed internally instead of iodides, but has no advantage over them and may disturb gastric digestion. It is probable that the potash and ammonia given in these drugs are of definite value in counteracting incipient acidosis due to the formation of organic acids of the fatty acid series which tend to diminish the alkalinity of the blood. Whenever the urine gives a port-wine colour with ferric chloride (the fallacy of the reaction with salicylates and other drugs being eliminated), due to the presence of diacetic acid, bicarbonate of sodium should be given in large doses so as to counteract this acidosis.

It is very important to prevent or remove gastro-intestinal catarrh,

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[1] Cavazzini. *Riv. crit. di clin. med.*, 1907, viii, 17.

unless syphilis can be excluded by
the Wassermann reaction

In Egyptian splenomegaly in which cirrhosis appears later, splenectomy before cirrhosis is sufficiently advanced to cause ascites or jaundice has been recommended (Day and Richards), but the condition is allied to Banti's disease & is not ordinary cirrhosis

Day and Richards. Trans. Soc. Trop. Med. and Hyg., 1912, V, 333.

Mayo, W. Ann. Surg., 1924, LXXX, 419. ~~[illegible]~~

LARRABEE. Amer. Journ. Med. Sc., Phila., 1924, CLXVIII, 54.

which by the production of toxic bodies aggravates the changes in the liver. This can be most satisfactorily obtained by keeping the bowels freely open by salines, such as sulphate of sodium and of magnesium, natural Carlsbad or other mild purgative waters, and by giving fractional doses ($\frac{1}{40}$ to $\frac{1}{20}$ grain) of calomel, which prevent fermentation without purging or interfering with digestion. Arsenic, salicylates, capsicum, and the tinctures and spirituous solutions of drugs should be avoided.

Spa Treatment.—In the very early stages of the disease considerable benefit may be obtained from a course at one of the numerous spas. The regulated life enjoined when taking the cure and the effects of mildly purgative water in reducing portal engorgement and diminishing intestinal catarrh and fermentation are factors for good in the spa treatment of early cirrhosis, but little real benefit is likely to result in the later stages of the disease, and the fatigue necessitated by the journey is likely to diminish the failing powers of the patient. The following spas may be mentioned :—Harrogate, Llandrindod Wells, Vichy, Vals, Ems, Neuenahr, Homburg, Wiesbaden, Baden-Baden, Kissingen, Carlsbad, Marienbad, Franzensbad, Tarasp.

Splenectomy.—In cirrhosis due to poisons manufactured in the spleen (*vide* p. 188) removal of the spleen is a logical, if heroic, form of treatment. Out of seven patients thus treated two died from the operation (Jullien [1]), there were 3 "hospital deaths" among W. Mayo's 10 cases, and one success in Lattabee's 2 cases.

Vid [illegible] Martigny [illegible]

2. **Palliative and Symptomatic Treatment.** — *Dyspepsia.* — The treatment of dyspepsia must depend on its nature ; if it is irritative and due to gastritis, bismuth, dilute hydrocyanic acid, bicarbonate of sodium, are indicated. When it is atonic and due to inactivity of the gastric mucous membrane, tonics, such as dilute nitrohydrochloric acid, quassia, liquor strychninae, should be taken after meals, and pepsin in various forms may be given with food. For the relief of flatulence intestinal antiseptics, such as guaiacol, creosote, thymol, salol, naphthalene tetrachloride, and salicylate of bismuth, are often recommended ; but it is better to give minute doses ($\frac{1}{40}$ to $\frac{1}{20}$ grain) of calomel twice or three times a day or saline purges.

Vomiting from gastritis should be treated by starvation for a time and by drugs, such as bismuth, bicarbonate of sodium, dilute hydrocyanic acid, or a little morphine. When food is resumed, after a few hours' rest, peptonised milk in small quantities should be given cold. The application of an ice-bag over the stomach has been thought to act as a sedative.

In the earlier stages *constipation* should be prevented by plenty of water, abdominal massage, and gentle exercise. Mild purgatives and laxatives should be employed if necessary, but powerful drugs, such as elaterium, should be avoided, as they may set up enteritis and exhausting diarrhoea. Salines, such as Epsom or Carlsbad salts ; a mixture of two drams each of sulphate of magnesium and of sulphate of sodium ; tartrate of sodium, or waters, such as Hunyadi János, Friedrichshall, Püllna,

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[1] Jullien. *Arch. prov. de chir.*, Paris, 1911, xx, 90.

Æsculap, Apenta, and Franz Josef, may be taken with advantage before breakfast. If these means are not sufficient, a blue pill may be taken overnight in addition, or recourse may be had to small doses ($\frac{1}{6}$–$\frac{1}{2}$ grain) of calomel, or to cascara, rhubarb, scammony, euonymin, podophyllin, iridin.

Early in the disease *diarrhoea* or looseness of the bowels may be due to chronic alcoholism; if this is troublesome, bismuth, aromatic chalk and opium mixture, dilute sulphuric acid, tannigen, or, if really necessary, the enema opii should be given. The diet should, however, be supervised in the first instance, and alcohol strictly tabooed. In the late stage, when toxaemia has developed, diarrhoea may set in and kill the patient. But when slight, it is not always advisable to check the diarrhoea, as it may serve a useful purpose, like diarrhoea in advanced renal disease, in diminishing toxaemia.

Pain over the liver or the minor discomfort of weight and fulness may be relieved by the application of leeches or dry cupping. When the liver is enlarged relief may be experienced from the application of a bandage to support the organ. Hepatic pain and discomfort may be connected with active congestion of the liver, and should be treated by saline purges and by a few 20-grain doses of chloride of ammonium.

The drowsy, semi-comatose condition, due to toxaemia from hepatic insufficiency, may be relieved or even removed by transfusion of saline solution into the muscles or into the veins, the former being the more suitable; the transfusion may be repeated after twenty-four hours. I have seen a patient so improved that he went out of the hospital well: six months later he died of lobar pneumonia; at the necropsy the liver weighed 88 ounces and was markedly cirrhotic. In another case life was prolonged for seven and a half weeks after ascites had disappeared. On the other hand, transfusion often fails or gives rise to transient improvement only. Copious enemas of water may be given, but are not very successful. When diacetic acid is present in the urine, bicarbonate of sodium (ʒij to the pint) should be added to the water used for transfusion, so as to counteract acid intoxication. Hot-air baths often make the skin act and sometimes relieve the drowsy, toxaemic condition. Perspiration may be much accelerated by giving a hypodermic injection of pilocarpine ($\frac{1}{8}$–$\frac{1}{6}$ grain) before the bath; its depressing effect may be prevented by combining it with liquor strychninae.

Portal engorgement manifested by hepatic enlargement and tenderness, ascites, dyspepsia, and delay in the absorption of food (*vide* opsiuria, p. 232), is treated by saline purges. The increased osmosis into the bowels diminishes the pressure in the portal vein. Sulphate of magnesium and sulphate of sodium combined may be given on an empty stomach, alone or after a dose of calomel. The good effects are shewn by a considerable diminution in the size of the liver.

Direct puncture of the liver has been occasionally, but usually unintentionally, performed. In a case, mentioned by Goodhart,[1] in which it was done accidentally in tapping, the woman, who was evidently dying,

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[1] Goodhart. *Guy's Hosp. Gaz.*, May 28, 1898.

was greatly improved by the withdrawal of blood from the liver. It is, however, dangerous, and should not be countenanced. Abstraction of blood from the spleen by means of a trocar has also been described as giving satisfactory results,[1] but it is dangerous and cannot be recommended. Massage to the abdomen, and especially to the liver, has been advocated to diminish the portal congestion which precedes haematemesis (Lecerf[2]).

Administration of Liver Substance.—Attempts have been made to compensate for the disturbance of hepatic function in cirrhosis by giving liver substance by the mouth, or injecting an extract of liver (hepatin) under the skin.

Mouras[3] collected 14 cases of cirrhosis treated in this manner, and though none could be regarded as cured, the results were encouraging; in 7 ascites disappeared and in the others improvement followed. The amounts of urine and of urea were increased, and the quantity of urobilin in the urine was diminished. In 26 cases collected by Gyr[4] in 1908 cure was reported in 13, a more optimistic estimate. Three or more ounces of pulped liver may be given daily. The only bad effect appears to be diarrhoea.

3. To Promote the Compensatory Mechanisms by means of which the Disease becomes Latent.—Nature's compensatory efforts in cirrhosis are (*a*) increase in the collateral circulation between the branches of the portal vein and the general systemic veins (*vide* p. 209) and (*b*) hyperplasia of the liver cells. These compensatory mechanisms have already been discussed in the section on Prognosis, and the operation for promoting vascular adhesions around the liver was described in the section on the Treatment of Ascites (p. 260).

Hyperplasia of the Liver Cells.—There is no drug, as far as is known, that can safely be employed for this purpose, though one would naturally turn to arsenic as likely, from its power of stimulating growth elsewhere, to have this effect. But it might set up gastro-intestinal irritation and autointoxication; and, indeed, there is reason to believe, both from increased frequency of ascites during the epidemic of arsenical poisoning in the north of England in 1899–1900 and from experimental results, that arsenic may cause cirrhosis. In the present state of our knowledge the administration of arsenic in cirrhosis is contraindicated. As was pointed out elsewhere, it is theoretically possible that the surgical production of vascular adhesions around the liver may improve the circulatory conditions and favour hyperplasia of the hepatic cells. It is hardly necessary to add that the general health should be improved as far as possible, and that fresh air, sun, and careful feeding are important factors in the treatment.

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[1] Remlinger. *Journ. des praticiens*, 1901, p. 659.
[2] Lecerf. *Thèse de Paris*, 1901.
[3] Mouras. *Thèse de Paris*, 1901.
[4] Gyr. *Rev. méd. de la Suisse Rom.*, Genève, 1908, xxviii, 413.

PIGMENTED CIRRHOSIS

In the description of the histological appearances of portal cirrhosis it was pointed out that pigmentation of the liver cells and of the fibrous tissue may occur under certain conditions, and that some slight pigmentation of the cells is not uncommon in ordinary examples of the disease. Pigmentation of a cirrhotic liver may, in exceptional instances, be due to foreign particles of carbon, stone, or metals reaching it by the bloodstream (*vide* Cirrhosis anthracotica). Some degree of pigmentation with haemosiderin, an iron-containing derivative of haemoglobin, may be seen in cirrhotic livers after local haemorrhages into the substance of the organ. In the foregoing instances the amount of pigmentation is comparatively small and of little importance. The more important forms of pigmented cirrhosis are (i) that occurring in haemochromatosis and bronzed diabetes, and (ii) malarial pigmentation of a cirrhotic liver.

Cirrhosis Anthracotica.—In this very rare condition cirrhosis of the liver is associated with the presence of small masses of carbon in the organ. It is analogous to anthracosis of the lungs (coal-miner's lung). Welch[1] described such a case, and Adami[2] speaks of a similar condition in relation to silicosis. The condition is probably not so rare as the paucity of recorded cases would suggest, as it may not be detected unless the liver is microscopically examined.

A chimney-sweep aged forty-seven was admitted to St. George's Hospital on June 4, 1892, with a transverse fracture of the left leg; a month later he had parotitis, and on September 8 he became jaundiced and ascitic; ten days later the abdomen was tapped, and on September 25 he died from asthenia. The trachea, lungs, and stomach were deeply pigmented. The liver (59 oz.) was cirrhotic, but not pigmented to the naked eye. Microscopically there was old multilobular cirrhosis with rather extensive pigmentation of the fibrous tissues. The pigment was black and amorphous, quite different from the yellow pigment

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[1] Welch, W. H. *Johns Hopkins Hosp. Bull.*, 1891, ii, 32.

[2] Adami, J. G. *Sajous' Annual*, 1898, ii, 313, article "Cirrhosis."

^| it is said to occur in charcoal-burners and leather workers (Lancereaux).

of haemochromatosis, and was probably soot conveyed by the portal vein to the liver. It is quite possible that such pigmentation of sweeps' livers in some degree is not uncommon. Alburger[1] described a case in a woman and suggested that the carbonaceous pigmentation depended on disease of the bronchial glands whereby their normal filtering action is impaired.

PIGMENTARY CIRRHOSIS OF HAEMOCHROMATOSIS.—*Synonym*: Diabète Bronzé.—**History and Etiology.**—Bronzed diabetes, or diabète bronzé, was first noticed by Troisier[2] in 1871, and was referred to by Trousseau in his clinical lectures. Hanot and Chauffard[3] in 1882 described two cases of diabetes with enlarged and pigmented cirrhotic livers. In 1886 Hanot,[4] in collaboration with Schachmann, described a further case and defined the condition as a morbid entity under the name "diabète bronzé." They took the view that the diabetes was the primary lesion, and that as a result the hepatic cell became stimulated to an increased production of pigment. Letulle[5] believed that as a result of hyperglycaemia the pigment was formed from the haemoglobin by the cells of various organs, and that it was stored up in the liver cells, though not produced there. In France the association with diabetes has been mainly insisted on. In Germany more attention has been paid to general pathological pigmentation without glycosuria. In 1889 v. Recklinghausen[6] described the condition of haemochromatosis, which consists in infiltration of the organs with pigment.

Opie[7] suggested that a toxin both causes haemolysis and acts on the cells of the liver and other organs so that they transform the soluble blood pigment reaching them into insoluble haemosiderin. Repeated injections of toluylenediamine into dogs have produced some pigmentation of the liver and other organs with an iron-containing body (Auscher and Lapique,[8] Meunier,[9] Biondi[10]); but the results are not comparable to haemochromatosis. Adami[11] and M. Abbott[12] regard bacterial activity as the cause both of the haemolysis and of the degenerative changes in the cells of the pigmented viscera, and suggest that it is a chronic intestinal infection. Examination of the blood, however, does not shew evidence of anaemia, haemolysis, or of haemolysins (Elmer[13]); and the bone marrow does not shew hyperplasia. Sprunt[14] suggests that haemochromatosis is a metabolic disease causing pigmentary degeneration of the cells followed by a reactive fibrosis of the organs, and not the result of blood-destruction. In the light of chronic haemolytic jaundice, the absence of jaundice in haemochromatosis militates against the existence of haemolysis. Potter and Milne,[1] who collected 51 cases of bronzed diabetes, consider that hepatic cirrhosis is the primary change and that there is an exaggeration of the siderosis often present in that disease (*vide* p. 207). There is not any evidence that suprarenal insufficiency is responsible for haemochromatosis.

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[1] Alburger. *Proc. Path. Soc.*, Phila., 1905, p. 137.

l.c/ [2] Troisier. *Bull. Soc. Anat.*, Paris, 1871, xlvi, 231.

[3] Hanot et Chauffard. *Rev. de méd.*, Paris, 1882, ii, 385.

[4] Hanot et Schachmann. *Arch. de physiol. norm. et path.*, Paris, 1886, 3. s., vii, 50.

[5] Letulle. *Bull. et mém. Soc. méd. d. hôp. de Paris*, 1897, 3. s., xiv, 205.

[6] Von Recklinghausen. *Tagebl. d. Versamml. deutsch. Naturf. u. Aerzte*, Heidelberg, 1889, S. 324.

[7] Opie. *Journ. exper. Med.*, 1899, iv, 279.

b/ [8] Auscher et Lapique. *Compt. rend. Soc. Biol.*, Paris, 1895, xlvii, 402.

[9] Meunier. *Thèse de Paris*, 1898.

[10] Biondi. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1895, xviii, 176.

[11] Adami. *Journ. Am. Med. Assoc.*, 1899, xxxiii, 1506.

[12] Abbott, M. *Journ. Path. and Bacteriol.*, Edin. and Lond., 1901, vii, 55.

[13] Elmer. *Interstate Med. Journ.*, St. Louis, 1911, xviii, 912.

[14] Sprunt. *Arch. Int. Med.*, Chicago, 1911, viii, 75; Journ. Exper. Med., N.Y., 1912, xvi, 607.

Pigmentation occurs in the cells of the liver, pancreas, and secreting glands, in the interstitial tissue of these organs, in the lymphatic glands, in the heart muscle, intestines, and least of all in the skin. There are two pigments. The cells and interstitial tissue of the liver, pancreas, secreting and lymphatic glands contain a ferruginous pigment, haemosiderin. Whereas in the muscular fibres of the intestines and heart, and sometimes in the liver (Potter and Milne), an iron-free pigment called haemofuscin is found. The iron-containing pigment is present in much larger amount than the iron-free pigment. The relation of these two pigments offers scope for discussion ; the iron-free may be only a later stage of the iron-containing pigment (M. B. Schmidt[2]), ~~or they may be independent~~. Goebel[3] found ~~this iron-free pigment~~ in the smooth muscular fibres of various organs in old persons, so it may be regarded as more or less physiological.

The pigmentation of the skin is due ~~to an iron-free pigment, apparently the same as the normal (Opie)~~. The muscular fibres of the heart contain both haemosiderin and haemofuscin. In the earlier stages of haemochromatosis the pigment accumulates in the cells of the organs, the liver being most affected. The general opinion as to the sequence of events is that as the cells become infiltrated with pigment, they progressively degenerate, undergo necrosis, and liberate the pigment, which passes into the interstitial fibrous tissue of the organ ; and that interstitial fibrosis of the liver and pancreas develops in the wake of the pigmentary change. It has, however, been thought that the pigmenta~~tion is secondary to the~~ hepatic cirrhosis (Potter and Milne). When the fibrosis of the pancreas reaches an extreme degree, with disappearance of the islands of Langerhans, diabetes results. Diabetes mellitus is therefore a late result of the haemochromatosis. The condition is not common ; in 1897 Letulle collected 30 cases, in 1906 Futcher[4] 35 cases, and 1911 Sprunt 50 cases of bronzed diabetes and 13 without diabetes.

A man aged fifty-two died in a drowsy state after paracentesis for ascites in 1902 under my care in St. George's Hospital. The liver was cirrhotic and of a greenish-blue colour. Multilobular cirrhosis with very well-marked haemo-

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[1] Potter and Milne. *Am. Journ. Med. Sc.*, Phila., 1912, cxliii, 46.

~~[2] Schmidt, M. B. *Virchows Arch.*, 1889, cxv, 397~~.

[3] Goebel. *Ibid.*, 1894, cxxxvi, 482.

[4] Futcher. *Am. Journ. Med. Sc.*, Phila., 1907, cxxxiii, 78.

and this view was confirmed on experimental grounds by Rous and Oliver.

, by union with the haemoglobin normally liberated,

From metabolism observations on 2 cases McClure found ~~There is~~ some evidence ~~that there is~~ of an abnormal retention of iron in the body ~~(McClure)~~.

Mallory, Parker, and Nye from experimental observation on rabbits and apes believe that a toxic agent, which they suggest is copper, leads to the formation in the liver cells of the primary pigment haemofuscin, which does not give the reaction for iron but on breaking down liberates haemosiderin which does. When the hepatic cells contain a certain amount of pigment, they necrose, and compensatory regeneration of the liver cells and secondary cirrhosis follow. The liberated pigment is picked up by the endothelial cells and so occupies the interstitial tissues.

Pigmentation begins in the endothelial cells lining the sinusoids and the liver cells, and after a considerable time appears in varying degree but almost synchronously in the cells of the pancreas, heart, kidney, cortex of adrenals, mesenteric glands, gastro-intestinal tract, and skin. In the liver and pancreatic cells haemofuscin readily changes into haemosiderin, but not so in muscle fibre. (Mallory, Parker, and Nye)

either to iron-free pigments haemofuscin or haemosiderin or to melanin from suprarenal disorder; these two forms of pigmentation usually occur separately, but they may be combined (Mills).

At the City Hospital, Boston, Mass. there were 10 cases in the year 1922-23, and 6 in the previous 24 years (Mills).

Rous and Oliver. Journ. Exper. Med., Baltimore, 1918, XXVIII, 629

McClure. Arch. Int. Med., Chicago, 1918, XXII, 610

Mallory, Parker, and Nye. Journ. med. Res., 1921, XLII, 461

Mills, S. E. Arch. Int. Med., Chicago, 1924, XXXIV, 292.

Among 16 cases at the Boston
City Hospital, Mass. there were
three in women (Mills).

may [illegible] [illegible]
of the [illegible] [illegible]

chromatosis was then found. During life there had been no glycosuria, and the skin, though somewhat earthy in colour, was not noticeably pigmented. The pancreas was not kept for microscopic examination, as the body was decomposed and no suspicion of haemochromatosis arose until after microscopic examination of the liver. In 1907 I saw in consultation a man aged sixty-six with ascites, an alcoholic history, diabetes, and diacetic acid in the urine; the skin was somewhat pigmented. The abdomen was tapped twice and he died comatose. At the necropsy there was old cirrhosis of the liver with marked haemochromatosis; the pancreas was much pigmented, and the intestines were of a leaden colour. Loehlein[1] has collected 3 cases of primary carcinoma in livers with cirrhosis due to haemochromatosis. I have seen extensive haemochromatosis with cirrhosis in a liver shewing secondary endotheliomatous growths (Fig. 72). In the museum of St. Bartholomew's Hospital there is a piece of liver shewing a simple cyst, which has the characteristic brick-red colour of the liver in haemochromatosis.

Probably pigmentation of the liver often assumed to be due to past malaria is in reality caused by haemochromatosis.

Age.—Haemochromatosis occurs chiefly between forty and fifty years of age.

Sex.—In Futcher's 35 cases of bronzed diabetes there were 33 males and 2 females; about the authenticity of the 2 female cases there is room for suspicion. Since then Roberts[2] described a case ^ in a woman. of bronzed diabetes In M. Abbott's case of haemochromatosis and cirrhosis in a woman diabetes had not appeared. ^

Morbid Anatomy.—The liver is ~~nearly always~~ enlarged, sometimes ^/ often considerably; the left lobe may be larger than the right. It has a striking deep-red or maroon colour, resembling that of brick dust or iron rust, and shews multilobular cirrhosis. As might naturally be expected, the pigmented liver is less permeable to Röntgen rays than a normal one (Jeanselme[3]). Microscopically there is a brownish-yellow pigment, which gives the reaction for iron with ferrocyanide of potassium and hydrochloric acid (Perl's test) in the liver cells, in the endothelial cells lining the vessels, in Kupffer's star-like cells, and in the fibrous tissue enclosing the lobules. The pigment collects first in the liver cells in the periphery of the lobules; as it accumulates the cell nucleus shews signs of degeneration, and eventually the cells necrose and break up. The pigment is picked up by endothelial leucocytes which collect in the lymphatics and mechanically damage the connective tissues so that proliferation of the fibroblasts follows (Mallory[4]). The hepatic artery shews endarteritis obliterans. The muscular coat and adventitia of the arteries and veins contain haemofuscin.

and often [illegible]

The *spleen* is ~~enlarged~~; firm, and pigmented/. The *pancreas* is enlarged, pigmented, and shews chronic interstitial fibrosis. The *suprarenals* ^ ~~do not shew any constant change~~. The *intestines* are pigmented from haemofuscin in the muscular fibres and haemosiderin in the epithelial cells of the glands.

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[1] Loehlein. *Beitr. z. path. Anat. u. z. allg. Path.*, Jena, 1907, xlii, 539.
[2] Roberts, M. *Brit. Med. Journ.*, 1911, ii, 1248.
[3] Jeanselme. *Presse méd.*, Paris, 1897, p. 58.
[4] Mallory. *Johns Hopkins Hosp. Bull.*, Balt., 1911, xxii, 71.

The **symptoms** may be those of cirrhosis or of diabetes (diabète bronzé). The signs are generalised pigmentation with enlargement of the liver and spleen. The pigmentation of the skin is chiefly on the exposed parts of the body, and its occurrence may perhaps be determined by sunlight (Potter and Milne) ; ~~it may be of~~ a slaty colour and might suggest the rare condition of argyria, in which the skin becomes permanently dis~~coloured as a result of the~~ medicinal use of silver salts. Generalised pigmentation is present in most but not in all the cases ; when it is absent the existence of pigmented cirrhosis is only discovered after death.

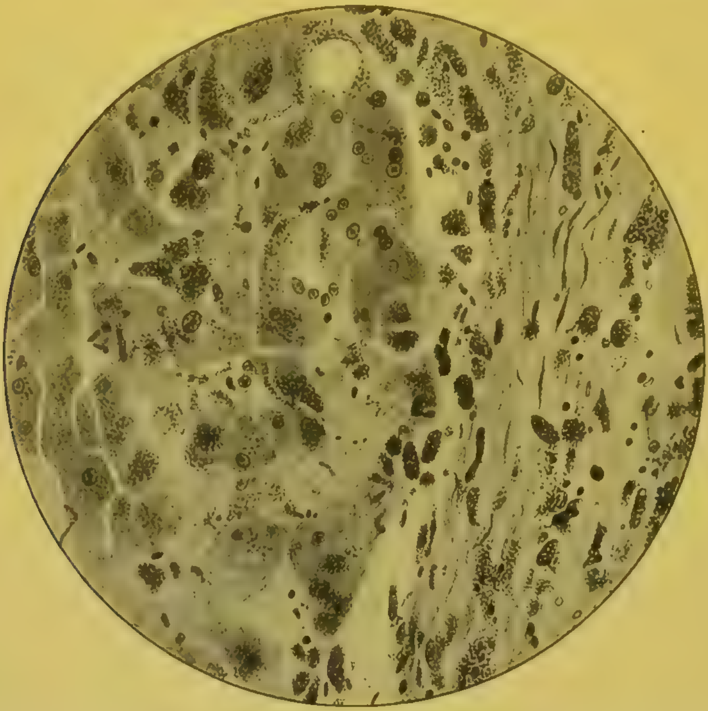

FIG. 38.—Pigmentation of the liver cells and fibrous tissue in cirrhosis of haemochromatosis. Some of the pigmented cells shew degeneration, and in some instances the outlines of the cells around the collections of pigment have gone. (From case described by Dr. Maude Abbott, who kindly provided me with some of the liver.) × 220.

The liver is usually, but not always, bigger than in health. ~~The enlargement of~~ the liver and spleen is usually progressive and relatively equal (Osler[1]). In a certain number of cases haemorrhages occur, and in a few instances there have been recurrent attacks of purpura, especially on the legs (Anschutz[2]). Abbott's case died of haemorrhage from an ulcerated oesophageal varix. T~~here may be ascites, which~~ has been found to contain sugar (Margain[3]), and the subcutaneous abdominal

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[1] Osler. *Am. Journ. Med. Sc.*, 1902, cxxiv, 766.
[2] Anschutz. *Deutsches Arch. f. klin. Med.*, Leipz., 1899, lxii, 411.
[3] Margain. *Rev. de méd.*, Paris, 1905, xxv, 214.

[illegible] according to [illegible] haemolysis or haemorrhage [illegible] (Mills) [illegible]

Jaundice was present in 9 of Mills' 17 cases.

The ~~[illegible]~~ liver and spleen were stated by Osler to be enlarged and to vary in size together, and by Mills not to be constantly enlarged nor to correspond in this respect.

Ascites was noted in 18 out of 75 cases (Brandon and Healy), and [illegible] in 8 out of Mills' 17 cases, and he [illegible]

Brandon and Healy. Arch. Int. Med., Chicago, 1921, XXVII, 406.

There is hyperglycaemia with or without glycosuria but no anaemia.

From pigmentation of the ductless glands pluriglandular insufficiency with desappearance of the secondary sexual characteristics has been described (Claude and Sourdel).

Removal of a piece of skin and the presence in microscopic sections of haemosiderin in the sweat glands or of haemofuscin in the smooth muscle fibres of the vessels wall assist in the diagnosis. The presence of intra cellular haemosiderin, pointing to renal siderosis is also of diagnostic value (Rous)

in the urine

Claude et Sourdel. Bull. et mém. Soc. méd. des hôp. de Paris, 1912, 3. s., XXXIV, 32

Rous, P. Journ. exper. Med., Baltimore, 1918, XXVIII, 645 veins may be dilated. There may be dyspepsia and pain on the right side of the abdomen. Diabetes is a late phenomenon and does not always supervene; when it does, it usually appears a year before death. It is severe in form, and is nearly always accompanied by acidosis. The onset may be somewhat abrupt, with weakness, diarrhoea, oedema of the legs, and may lead to death from coma or from destructive lung changes.

Diagnosis.—Signs of cirrhosis, glycosuria, and pigmentation of the skin justify a certain diagnosis. In cases of cirrhosis with cutaneous pigmentation, but with no glycosuria, the disease may be suspected, but unless the pigmentation is very considerable cannot be positively diagnosed, since a dirty earthy colour is not uncommon in ordinary cirrhosis, and past jaundice may discolour the skin. As bearing on the question of diagnosis it may be mentioned that diabetes occurs in 2 per cent of all cases of cirrhosis (Potter and Milne), that cirrhosis occurs in 5 per cent of all cases of diabetes (Simmonds[1]), and that Futcher noted bronzing twice in 256 cases of diabetes.

Differential Diagnosis.—From *Addison's* disease the enlargement of the liver and spleen and glycosuria should distinguish it. In some instances of *hypertrophic biliary cirrhosis* the colour of the skin may resemble that of haemochromatosis, but the existence of jaundice as shewn by the conjunctivae should prevent a mistake. In *chronic splenic anaemia* there is sometimes cutaneous pigmentation, possibly from the effects of arsenic given medicinally, and some resemblance to haemochromatosis without glycosuria might result. The spleen is greatly enlarged in splenic anaemia, and only moderately in haemochromatosis, while the course of the disease is usually prolonged in splenic anaemia.

In most exceptional cases alcaptonuria may be associated with remarkable pigmentation of the face, ears, and internally of the costal cartilages (*ochronosis*, Virchow). In these cases the reduction of copper by the urine might suggest bronzed diabetes; further examination of the urine would, however, prove the absence of sugar.

Prognosis is bad. Life may be prolonged for years and death may occur from gastro-intestinal haemorrhage as in cirrhosis. When diabetes appears, the fatal termination is usually not delayed beyond a year.

Treatment.—If there is diabetes, the treatment should be directed to that condition by appropriate dieting, opium, and alkalis. Before the onset of glycosuria the treatment should be the same as that of early cirrhosis, viz. a bland diet and drugs directed to prevent intestinal fermentation and putrefaction.

MALARIAL PIGMENTATION OF A CIRRHOTIC LIVER.—In the past malaria was regarded as an established cause of hepatic cirrhosis, and several forms of malarial cirrhosis were described. Frerichs, Lancereaux, Phillips,[2] and Kelsch and Kiener[3] recognised malaria as a cause of hepatitis, and the two latter described in great detail the changes induced in the liver by chronic malaria. In one form, called "hyperhémie phlegmasique," the liver is large, shews perihepatitis, cloudy swelling of the liver cells, and small-celled infiltration of the portal spaces. In another form, nodular parenchymatous hepatitis, the liver is large, soft, and contains small nodules composed of liver cells undergoing hyperplasia; cirrhosis may subsequently develop. Kelsch and Kiener believed that the fibrosis was due to metaplasia of the liver cells, but the view that connective tissue is formed from epithelial cells is entirely opposed to modern pathological teaching. The resulting cirrhosis is described as either unilobular or multilobular. Both the fibrous tissue and the liver cells are infiltrated with pigment.

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[1] Simmonds. *Berlin. klin. Wchnschr.*, 1909, xli, 531.

[2] Phillips, L. P. *Records of the Egyptian Government School of Medicine*, 1904, ii, 47.

[3] Kelsch et Kiener. *Arch. de physiol. norm. et path.*, 1878, p. 571; 1879, p. 354.

As Barker[1] points out, the degenerative and necrotic changes in the liver cells produced by severe malarial infection are exactly the conditions favourable to the production of chronic fibrosis of the liver. Further, in malaria there may be considerable gastro-intestinal disturbance which might lead to cirrhosis of the liver, and it has also been thought that the production of poisons in the spleen might cause secondary cirrhosis (Chauffard,[2] *vide* p. 188). Barker suggested that the pigmentation of the liver cells, which is most marked in the peripheral parts of the lobules, may act as an irritant and set up fibrosis of the organ, in the same way that the inhalation of carbon particles induces pneumoconiosis. Osler,[3] on the other hand, says that during fifteen years' practice at the Johns Hopkins Hospital there was no case of advanced cirrhosis due to malaria. Welch is quoted by Barker as having seen only one case of malarial cirrhosis in New York, and that in an Algerian.

Without denying the possibility that the necrotic changes in the liver cells induced by malaria may be succeeded by cirrhosis, this sequence of events is evidently so rare that when cirrhosis occurs in a malarial subject the question must arise whether it is cirrhosis caused by malaria or merely cirrhosis in a malarial patient. Alcohol and intestinal toxaemia may be potent causes of hepatic cirrhosis in patients suffering from chronic malaria, or a patient with latent cirrhosis may contract malaria. In either of these events the liberated blood pigment may be deposited in the liver and produce a pigmented cirrhosis. Clinically there is nothing very special in cirrhosis in a malarial subject. The signs are those of ordinary portal cirrhosis terminating with ascites. From the influence of malaria the spleen is more markedly enlarged than in ordinary portal cirrhosis.

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[1] Barker. *Johns Hopkins Hosp. Rep.*, 1895, v, 221.
[2] Chauffard. *Semaine méd.*, Paris, 1899, xix, 177.
[3] Osler, W. *Practice of Med.*, p. 557, 5th ed., 1905.

Jonesco and Popper describe portal cirrhosis of
splenic origin with hyperplasia of Kupffer's
cells which are loaded with malarial pigment.

Jonesco et Popper *Presse méd.*, Paris, 1922, 476